HUMAN LUMBAR FACET JOINT SYNDROME : CLINICAL AND PATHOLOGICAL FINDINGS

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ABSTRACT

As the only synovial joints in the spine, facet joints accompanied with the intervertebral disc bear a significant amount of stress from the body. As a result, degeneration of these structures will follow. The lack of knowledge in the significance of facet joint pathology in the pathogenesis of low-back pain overshadows the clinical syndrome resulting from facet joints. However, in order to investigate further the effects of the different modalities of conservative treatment in patients with conventional facet joint syndrome, we develop a new "scoring system" for assessing the progress of patients after treatment. In addition, cadaveric study for the morphological observation of articular cartilage in facet joints further delineates the relationship between the severity of degeneration of articular cartilage and sex, age, level of the facet joint, side, superior or inferior facet, biochemical composition (water and proteoglycan) of the cartilage.

65 patients were involved in this clinical study. They were randomly arranged for manual or ultrasound therapy. The "score" of each patient was attained after calculation from the conventional criteria of facet syndrome, and the "score" between the initial and the "score" from each follow-up were compared.

240 facet joints and hence 460 articular surfaces from 24 cadavers were examined separately. The degree of degeneration from each articular surface was denoted by the introduction of "Gross Morphological Index", which was calculated from the percentage of different gradings on that articular surface. The validity of the gross observation was confirmed by histological sections of randomly selected articular surfaces. Pooled cartilage from different grades was studied. The water content and proteoglycan content inside the cartilage matrix was measured.

Patients with facet syndrome were usually younger age group housewives with chronic presentation. The lower part of lumbar spine was principally affected. The significant decrease of "score" with increasing frequency of physiotherapy treatment had no correlation with type of treatment given (whether ultrasound or manual therapy), sex, age or occupation. However, the decrease of "score" showed a highly correlation with the initial score, duration of physiotherapy treatment, and duration of symptoms. The decrease of "score" was the same for patient with higher or lower initial score. For chronic patients, the "score" is always higher than for the acute cases. Nevertheless, with increasing frequency of treatments, the decrease of "score" was more pronounced for acute cases.

The morphological changes of facet joint cartilage were compatible with the known picture of osteoarthritis in other synovial joints. There was a high correlation between the macroscopic and microscopic examination. The frequency and degree of osteoarthritic changes as reflected by the increase in "Gross Morphological Index" on the facet joint cartilage with increasing age. There was a higher degree of degeneration in older females than males. The L45 level showed a lower degree of degeneration, while the other 4 levels showed a higher degree of degeneration. No correlation was found between the degree of degeneration and the left or right side, nor between the superior and inferior facet. Facet cartilage with moderate degeneration showed a significant increase in water content compared with the normal cartilage. No significant difference was found when comparing the severely degenerative cartilage with normal or moderate degenerative cartilage. In addition, proteoglycan content of advanced degenerative cartilage showed a significant decrease when compared with normal or moderate degenerative cartilage.

The present postmortem study is not able to show any correlation between degeneration and the clinical symptoms. However, the higher incidence in younger

patients with facet syndrome and those older age group with higher degree of degeneration of facet joint cartilage in cadavers suggest that the symptoms may not correlate well with pathological changes. These findings are consistent with the poor correlation between radiological observations of facet joints and clinical symptoms. Furthermore, the similarities between the morphological, biochemical changes in facet joint cartilage and the other synovial joints would encourage further investigation into the effect of biomechanical stress on these joints in spine.

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CHAPTER ONE - INTRODUCTION

Low-back pain is a major medical disorder which significantly affects the general population. From 50% to 80% of the adult population in Western countries have at some stage of their life experiences low-back pain (1). In many industrialized countries the incidence and prevalence of low-back pain is increasing (2,3). Local epidemiological study on low-back pain also demonstrated that low-back pain has an incidence of about 40% among nurses and industrial area workers (4). Many studies (5,6,7) have demonstrated a clear-cut relation between such factors as heavy lifting, high physical activity at work, prolonged time away from work and psychological factors with the prevalence of low-back pain. Only a few papers (8) mentioned about the correlations between the severity of the pain symptoms and those causative factors, such as heavy duty work. Our recent study (see Appendix 1) revealed that the various causative factors, such as sex, age, anthropometric measurements, exercise frequency, smoking, past injury, work-relating factors (type of work, weight lifting frequency, bending or squatting frequency during work and so on) were not useful in predicting the severity of symptoms (mild, moderate and severe categories) among the Chinese industrial population. However, the outcome of low-back

pain is usually favourable. Nearly 90% were back to work within six weeks, 60% within one week.

Among the various diagnoses of low-back pain, arthropathy of the lumbar facet joint has long been recognized as an important source of low-back pain. With the classic work of Mixter and Barr (1934) (9), the role of the herniated intervertebral disc overshadowed the importance of facet joint disorders as a source of low-back pain and sciatica. However, facet syndrome constitutes a significant proportion of low-back pain (10). Interest in the lumbar facet joints has been revived in recent years, and various modalities of treatment for lumbar facet syndrome have been described by numerous authors (11,12,13,14). Nevertheless, the clinical criteria for making the diagnosis of lumbar facet syndrome are ill-defined. Some even question its existence. Consistent clinical criteria are essential for the accurate diagnosis and for predicting responses of the lumbar facet syndrome to a given treatment modality. We have utilized the existing criteria to diagnose facet syndrome and introduced a new scoring system for assessing the progress of patients after treatment, and compared the results between manual therapy and ultrasound therapy. In addition, we have delineated factors that have a predicting value for the outcome of the patients in the course of low-back pain.

The discussion on the morphological and biochemical background of low-back pain has been to a larger extent focussed on the lumbar intervertebral disc. To extend our knowledge of pathophysiological mechanisms capable of explaining low-back pain, it was believed that pathological changes (either morphological or biochemical, or both) of other lumbar structures such as facet joints, ligaments, muscles, will have a significant contribution. Although the role of articular cartilage in the pathogenesis of osteoarthritis is unclear, many authors still believe that the earliest osteoarthritic changes starts from the articular cartilage. However, there is a wide discrepancy between radiological signs of the disease and clinical symptoms in facet syndrome (15,16,17,18). But it has been reported that both the histology and the biochemical composition of proteoglycan of the cartilage (hip, knee or other articular cartilage) change with the severity of the osteoarthritic disease (19,20,21,22). Much works had been done for the biochemical changes in normal or degenerate articular cartilage in other joints. However, surprisingly little is known about their compositions in lumbar facet cartilage. Does the facet joint cartilage behave in the same way as the other articular cartilage morphologically and biochemically as far as degeneration is concerned? The objectives of my study are, firstly, to develop a

system for the morphological observation of articular cartilage in facet joints. Furthermore, using this new system, we try to find out the relationship between the severity of degeneration of articular cartilage and sex, age, level, side, superior or inferior facet, biochemical composition (water and proteoglycans) of the cartilage.

In summary, the main aims of my study are, firstly to delineate factors which have predicting value for the outcome of patients with "facet syndrome" in the course of low-back pain after physiotherapy treatment. Secondly, to find out the relationship between the severity of degeneration of facet joint cartilage from cadavers with sex, age, level of lumbar spine, sides, superior or inferior facet, biochemical composition (water and proteoglycans) of the cartilage.

CHAPTER 2 - LITERATURE REVIEW

2.1. Low-back pain and "Facet joint syndrome"

2.1.1. Cost of low-back pain

The annual prevalence of low-back pain has been reported to be anywhere from 5 to 25 percent and the incidence from 30 to 90 percent (3,7,23,24,25). However, a study shows that 85 to 90 percent of patients with attacks of low-back pain will have recovered within 8 to 10 weeks (26). Therefore, it is a self-limiting disease, but those 10 to 15 percent of back pain patients out of work for more than 8 weeks account for about 80 percent of the cost (23,27).

2.1.2 . Low-back pain in industry

In the United States, it has been estimated that about 2% of industrial workers injure their backs each year. However, the annual rate varies greatly ranging from less than 1% to greater than 15%, among different industries and industrial establishments (1). Truck drivers experience low-back pain more frequently than any other occupational group (1). In addition, workers exposed to heavy manual handling tasks are three times more likely to develop compensable low-back pain than

other workers (1). Only few studies did mention about the relationship between the severity of subjective back pain and the working load of the job (8,28). Among the 220 lumberjacks employed in felling work and 98 persons involved in light work, there were no statistically significant differences between these two groups with regard to the occurrence of low-back pain and the subjective degree of low-back pain.

2.1.3. Differential diagnosis of low-back pain

An exact definition of idiopathic low-back pain is difficult to determine. Utilizing the best medical evaluation, it is often impossible to determine the cause of the back pain precisely. Estimates of the proportion of all low-back pain that has no definite etiology range widely from about 20 to about 85 percent, depending in part on whether degenerative changes in the intervertebral spaces seen roentgenographically are included in the category of idiopathic low-back pain (1). However, in a small proportion of patients, a precise diagnosis can be made. Therefore, the primary aim in managing low-back pain is to identify these cases amongst the enormous number of back pain patients.

The following table shows the principal causes of low back pain and sciatica (29).

1.	Non-specific low-back pain	Facet joint syndrome Sacroiliac joint syndrome Piriformis syndrome Maigne's syndrome
2.	Structural/degenerative:	Lumbar disc protrusion Lumbar spondylosis Spondylolisthesis Lumbar canal stenosis Senile ankylosing hyperostosis Primary generalized osteo-arthritis
3.	Congenital/developmental:	Lumbo-sacral anomalies Scheuerman's disease Spinal cysts Root malformations Redundant cauda equina roots
4.	Abdominal disease	Direct involvement of spine Referred pain from viscera
5.	Vascular disease	
6.	Spinal tumours	
7.	Spinal inflammatory disease:	Pyogenic infection Tuberculosis Ankylosing spondylitis Brucellosis
8.	Bone disease	Osteoporosis Osteomalacia Paget's disease
9.	Trauma	
10.	Psychological causes	

Table 1. Causes of low-back pain and sciatica.

It seems reasonably that most of the residual majority cases originate from the interaction of degenerative change and the strains of everyday use of the back. Nevertheless, management of low-back pain could be successful only if treatment is directed to the pain-producing structures. Therapy directed toward a nonspecific diagnosis may lead to failure, and in many cases it may cause further damage. A retrospective study of 1293 cases of low-back pain treated over a 12-years period revealed that sacroiliac joint syndrome and posterior joint syndrome (facet joint syndrome) were the most common referred-pain syndromes, whereas herniated nucleus pulposus and lateral spinal stenosis were the most common nerve root compression lesions (10). Referred pain syndromes occur nearly twice as often and frequently mimic the clinical presentation of nerve root compression syndromes. It also claims that if a specific diagnosis is established, it should yield excellent results in 84% patients.

2.2 Lumbar facet joint syndrome

2.2.1. Historical review

The lumbar facet joints could be a source of back pain was first suggested by Goldthwait (1911) (30) and reinforced by Putti (1927) (31). Putti concluded that

sciatic pain was commonly symptomatic of vertebral arthritis 'except in those rare cases in which it is a symptom of a neuritis of specific nature'. The term 'Facet syndrome' was first used by Ghormley (1933) (32). He pointed out that many of the aches and pains known as backache or true joint pain represented the same type of joint pain seen in arthritis of other synovial joints. Badgley (1941) stressed the importance of the facets in low back and leg pain, feeling that 80% of low-back pain and sciatica were due to referred pain and not direct nerve irritation (33). Mixter and Barr (1934) introduced the concept of the rupture of the intervertebral disc as the etiology of low back and leg pain (9). Pedersen (1956), returned attention to the facet joint as a source of pain (34). Hirsch (1963) confirmed this by injection of 10% normal saline in the region of the facet joints, there was production of pain in the back and upper thigh (35). Mooney and Robertson (1976) found in 100 patients treated with injections of the facet joints that 62% had completely relief immediately after injection and 52% had at least complete or partial relief 6 months after the block (13).

The above findings suggested that the lumbar intervertebral disc is not the exclusive source of chronic low-back pain and sciatica; and that structural, degenerative, or inflammatory disease of the facet joints

may represent a major cause of low-back pain with or without sciatica (36).

2.2.2. Anatomy of the lumbar facet joint

2.2.2.1. Synovial joint

The adjacent vertebral bodies are bonded by the intervertebral disc anteriorly and by the apophyseal(facet) joints posterolaterally. The facet joints are formed by the superior and inferior articular processes of consecutive vertebrae. The facet joints are true synovial joints with hyaline cartilage surfaces and a joint space enclosed by a fibrous capsule lined with synovial membrane. Lewin studied extensively on the morphology of facet joints (37). He stated that the cartilage in normal facet joint was thickest over the centre of each facet, rising to a height of about 2 mm. Furthermore, 4 zones (tangential, transitional, radial, calcified) could be demonstrated under histological observation, which is similar with the other articular cartilages. The capsule of the facet joint combines with the ligamentum flavum on its medial and superior aspects, which takes the place of a true joint capsule. The outer components of the ligamentum flavum may prevent the capsule from being nipped between the two articular surfaces during movement and from protrusion into the intervertebral canal. The outer aspect of the

articulation is covered by a fibrous layer. A tiny hole is situated in the superior and inferior poles, which permits the passage of fat from the capsule to the extra-capsular space (38). The synovial lining is made up of villi that may vary in size, shape, appearance and which contain a rich supply of blood vessels.

2.2.2.2. Nerve supply

Each medial branch from the same level dorsal rami of spinal nerve supplies the facet joints above and below its course (38,39,40,41). Recent studies have demonstrated that each facet joint receives an additional innervation ventrally, from the dorsal ramus in front of the joint (42,43). Each joint, therefore, receives a multiple innervation: from a dorsal ramus, and two medial branches. Histological studies have shown that capsules of lumbar facet joints are profusely innervated with encapsulated, unencapsulated and free nerve endings (40,44,45), and are, therefore, awarded with the appropriate sensory apparatus to transmit proprioceptive and nociceptive information.

2.2.3. Functions of facet joint

The main function of all synovial joints is presumably to guide and stabilize flexion and extension movements. In lumbar facet joint, it also prevents

forward displacement and rotatory dislocation of the joints with the help of the orientation and the shape of facet joints. Besides, intervertebral disc as the principal weight-bearing components of the lumbar spine, the role of facet joints in weight-bearing is still controversial (46,47).

Adams and Hutton (1980, 1983) have demonstrated that under the conditions of erect sitting the facet joints are not impacted and bear none of the vertical load on the intervertebral joints; but in prolong standing with a lordotic spine, the impacted joints at each segmental level bear an average of some 16% of the axial load (48a,48b). The lower joints (L3-4, L4-5, L5-S1) bear a relatively greater proportion (19%), while the upper joints (L1-2, L2-3) bear less (14%) (48a). On the other hand, extension of the spine would involve downward movement of the inferior articular process of facet joints, and maximal pressure is detected on the inferior medial portion of the facets (49). Therefore, if a load continues to be applied to an extended joint, the upper vertebrae would rotates on the impacted inferior articular process or the opposite inferior articular process swings backwards straining its joint capsules. These would result the joint capsule being caught between the bones and injured (48b) or the top of the inferior articular process may irritate or erode the periosteum of

the lamina (50). Due to the lordosis of the lumbar spine gravity, it will tend to displace ventrad the vertebrae below the vertex of the lordosis. Since L3 constitutes this vertex, therefore, it has been hypothesized that the joints on the L4-5 and L5-S1 levels at least in part prevent such displacement (51). Moreover, the frontal inclination of the facets and the dorsally wedge-shaped disc of the L5-S1 segment is thought to suffer greater stress than the other synovial joints of the lumbar spine.

2.2.4. Pathophysiology and pathology of facet syndrome

At any one level, the motion segment is made up of three distinct parts: the two facet joints and the intervertebral joints(the disc). In the normal motion segment, the three joints are anatomically linked and mechanically balanced. Any trauma or pathological changes in one part of the complex will, in time, consequently spread to the two others through changes in the mechanical behavior of the construct. Thus, when the disc degenerates there is an increase in facet load (49). As the movements between the spinal segments become uneven, excessive, and irregular, this will cause dysfunction in facet joints with following degenerative changes.

Kirkaldy-Willis (1982) proposed a 3 stages theory for interpreting the pathogenesis of facet syndrome and degenerative changes in any other synovial joints (52). The rationale is that the human body will tend to stabilize an unstable joint by increasing the surface area of a joint where damage to the cartilage surface interferes with optimal function or by immobilizing the joint by the natural splintage of muscle spasm. The variety of degenerative changes in the motion segment can be divided into three phases that widely overlap (53,54).

1. Phase of dysfunction

Either acute trauma or repeated microtrauma to a primary restraint structure can involve the capsule, the synovium, the cartilage surface, or supporting bone. More often it primarily involves the capsule and synovium. The early joint response to trauma is synovitis. Chronic synovitis and joint effusion can inflate a traumatized capsule. The inflamed synovium may, in turn, project folds, which may become captured in the joint between the cartilage surfaces, and may in turn activate cartilage damage. In this phase, facet pathology will exhibit itself in the facet syndrome.

2. Phase of instability

As the changes continue in the facet joint, capsular stretching and progressive loss of joint cartilage will

progress to permanent laxity of the capsules. This increasing laxity will allow joint subluxation. Disc herniation is found in this phase. As a result, dynamic degenerative spondylolisthesis is found when laxity predominates in the posterior restraining structures and dynamic degenerative retro-olisthesis is found when laxity predominates in the disc (55). Both can produce dynamic lateral or central nerve entrapment. This has been confirmed experimentally (56,57).

3. Phase of restabilization

As the unstable of either or both facet joint and disc become increasing, attempts at restabilization will take the form of subperiosteal bone or bone formation along ligaments and capsule fibres resulting in perifacetal and peridiscal osteophytes and traction spurs. The facet joints surface becomes widen both in its ventral and dorsal aspects. The above changes will subsequently reduce movement and produce a stable motion segment. In this phase, both dynamic degenerative spondylolisthesis and/or retro-olisthesis become fixed and can produce symptomatic single-level spinal stenosis. Enlargement of facet joints and circumferential osteophytes around the disc space can also produce symptomatic single-level central and/or lateral stenosis.

2.2.5. Diagnosis of facet syndrome

2.2.5.1. Signs and symptoms

No specific sign or symptom could be responsible for the diagnosis of facet syndrome. Furthermore, the sensitivity of clinical examination of nerve root compression by herniated disc is about 77%, 23% of the cases are misdiagnosed. The specificity of clinical examination for the diagnosis of disc herniation is 90%, some 10% cases prove to be due to other cause, such as facet osteophyte, spinal stenosis or epidural varices. In fact, there is no scientific evidence that permits any claim that certain pain patterns are characteristic of these syndromes. Nevertheless, it is widely believed that the facet joints are a principal cause of low-back pain (10,33). Symptoms of a typical facet syndrome are: hip and buttock pain; cramping leg pain, primarily above the knee; low-back stiffness, especially in the morning or with inactivity or prolong static posture; and absence of parathesias. The typical signs are: local paralumbar tenderness; pain on spine hyperextension or lateral flexion; absence of neurologic deficit; absence of signs of nerve root tension; and hip, buttock, or back pain on straight leg raising test (58). Patients with facet syndrome usually will not present all the above symptoms and signs, also they may present with symptoms or signs

that may be indicative of herniated disc if these pathologies happen concomitantly. Some studies concluded that following the above criteria, their treatment protocol seems to be satisfactory (10,59,60).

A scoring system was formulated by Helbig and Lee in order to better the diagnosis criteria for lumbar facet syndrome and for predicting treatment response to facet joint injection (59). The scoring system has a total of 100 points, allocated as follow: back pain associated with groin or thigh pain, 30 points; corresponding radiographic changes, 20 points; and pain below the knee, -10 points. A score of 60 points or more indicates a very high probability of satisfactory response to facet joint injection (in their study claims to have 100% prolonged response) compare with 50% prolonged response after injection when only the above conventional criteria were used.

2.2.5.2. Other investigations

Plain radiography has little value as a diagnostic tool in low-back pain (59), while electromyography, myelography and CT scanning are of relevance only in nerve root compression syndromes. In a study of 100 patients, CT scans showed facet joint arthropathy in many patients who had normal plain films (61). Therefore, for

conditions in which pain is the only complaint, and no objective neurological signs is detected, other investigations are required. The diagnostic blocks and provocation radiology are the mainstay for the diagnosis of lumbar pain in the absence of neurological signs. The rationale is based on that if a structure is the cause of pain, then stressing that structure should reproduce the pain, and anaesthetising the structure should relieve the pain. Therefore, facet joints suspected of being the source of pain can be infiltrated with local anaesthetic, and relief of pain indicates the injected joint as the source (13,60). Although subject to certain technical limitation, and they do not reveal the actual cause of pain, these techniques are the only available means of objectively establishing at least the anatomical location of the cause of pain.

2.2.6. Treatment of facet syndrome

2.2.6.1. Conservative

1. Manual therapy

As a major treatment modality for facet syndrome, manual therapy seems to offer only shortlasting (1 to 3 hours) relief of pain an effect resembling certain drugs (62,63,64). But some studies still revealed a high percentage of excellent result after treatment (10).

Maitland (65) claims that synovitis or any intra-articular mechanical inflammatory process will present as a pain-through-range situation. If the situation is treated by passive movement techniques, then the initial selection of techniques and progression of treatment are under the heading of treating 'pain'. The facet joint can be responsible for patches of referred pain without there being any pain in the boundary of the facet joint. If the arthritic facet joint which is responsible for a patch of referred pain is stressed, it will be painful locally and, sometimes, reproduce the referred pain. Selection and progression of techniques are equivalent with those described for treating the pain with stiffness group. Nevertheless, there are two further ways in which techniques may be advanced. First way of progressing when mobilization has reached the limit of its effectiveness, manipulation of the kind that is localized or emphasized at the one faulty intervertebral level should be selected. It must stretch the facet joint and should be followed by repeat of the end-of-range mobilization, using both small and large magnitude movements. Second way of progressing when the symptoms are believed to be arising from an intra-articular disorder yet there is no synovitis, the initial progression of techniques is the same as previously. But when such mobilization has discontinued to produce an acceptable rate of progress

the technique selected must move the facet joint through a large amplitude while its opposing surfaces are compressed together. During the execution of the technique, if the right selection has been made, the patient will be conscious of local discomfort. A substantiated examination procedure is that, if the movement is continued, but the compression is gradually reduced, until minimal distraction is applied, the local discomfort goes (66).

2. Ultrasound therapy

No study has investigated the effect of ultrasound therapy or other heating devices in patients with facet syndrome, it is likely that this treatment, same as the other forms of treatment have failed to demonstrate any significant effect on the natural history or on return to work. Therefore, the short-termed and long-termed effect of the above therapy need to be investigated further. The exclusive deep heating effect by ultrasound has been shown that deep structures, such as the hip joint, can be heated (67). Dyson and Pond described the generation of heat, micromassage and alteration of membrane permeability, and experimental stimulation of tissue generation, and the reversible effect of blood cell flow with ultrasound therapy (68). Of all the effects of heat have been discussed, it is likely that the two most important in the treatment of pain are its psychological

effects and its counterirritant effects. The other physiological effect do exist, yet, in most clinical situations, the use of heat does not cause a significant rise in temperature in the structure where the pain is originating.

3. Other treatment modalities

As the pain associated with facet arthropathy is mediated by triple innervation, early attempts used surgical and later chemical means to destroy the nerves. Facet joint injection with steroids and lidocaine have been used as a diagnostic and therapeutic modality. Arthrography of facet joints has been used to study patterns of referred pain. Mooney and Robertson reported that 62 of 100 patients obtained relief from facet injection (13). In the series of injection performed by Fairbanks and co-workers, 56% of patients also obtained relief (69). Most recent study by Lewinneck and Warfield concluded that 75% of the patients had an initial response to injection, but only 33% were still pain-free after three months (70). Rees developed a procedure of bilateral rhizolysis in 1971, claiming a 99.8% success rate for 2000 operations (71). Subsequent studies, however, have questioned the validity of his results. Shealy used a temperature-controlled, radio-frequency cautery technique for denervation the facet joints (72). Other

Other studies of the surgical anatomy of the facet joint seriously question whether or not a true denervation was carried out.

2.2.6.2. Operative

Spinal fusion:

If many cases of low-back pain are definitely due to arthritis of facet joints, then the reasonable treatment in severe cases would be fusion of the lumbar spine. This has indeed been advocated and have been said to produce good results (33). Recent studies revealed that relief of symptoms was seen in patients with characteristic facet syndrome and significant disability by local spinal fusion (73). They also noted that the histologic changes seen in the excised facet joint's cartilage resemble those seen in osteoarthritis of other joints and chondromalacia patellae.

2.2.7. Prognosis of facet syndrome

The natural history of all low-back pain patients is favourable, that 6 weeks 90% were back to work, 60% within 1 week. No study has investigated the natural course of facet syndrome with or without treatment given, although it is difficult in clinical practice. A study has mentioned that some non-specific signs and symptoms may be of predictive value for treatment response to

facet joint injections (59). Warfield in his study on facet joint injection concluded that the only factor that seemed to correlate with response to facet injection was the presence of facet joint degeneration on x-ray (60). The onset, distribution of pain, response to anti-inflammatory, presence of positive straight-leg raising, and pain on extension did not correlate well with the response to treatment. Recent study by Bough, who investigates the value of facet joint arthrography with emphasis on the symptoms produce as a screening procedure prior to local spinal fusion (74). He concluded that the production of symptoms during facet arthrography is of little value as a screening procedure.

2.3. Articular cartilage of lumbar facet joints

Ostoearthritis can be summarized as deformations in which there is deterioration and mechanical loss of the articular surface associated with a disturbance of the configuration of the joint related to a series of reparative phenomena, which result in proliferation of new articular tissue at the margins and base of the joint (75). The sequence of these events and their mechanisms are not clearly established (76). Two broads but distinct mechanisms may be involved. One postulates that osteoarthritis begins as an intrinsic senescent or other degeneration of articular cartilage, while the other

states that osteoarthritis is caused by abnormal mechanical stress acting on joints. In reality both are involved. Although articular cartilage plays a major and central role in the etiology, pathogenesis, and clinical symptom complexes that are associated with the various forms of osteoarthritis, extracartilaginous structures figure quite prominently in aspects of the disease and clearly deserve more extensive study. These include sclerosis, necrosis and cyst formation of the subchondral bone; the formation of osteophyte; synovial hypertrophy, synovitis and fibrosis. They are present at different stages of degenerative processes.

2.3.1. Function of articular cartilage

Articular cartilage is a material which consist of a mixture of substances, the principal components of which are collagen fibrils and a hydrated glycoprotein gel. Therefore, the main function is transmission of loads and as a bearing surface. While the functions of the individual components of articular cartilage may be speculative, it is clear that cartilage as a whole serves to transmit loads and allow repetitive joint motion without breakdown. Its ability to deform under load also achieves greater affinity between the apposing sides of the joint, spreading the load over a greater surface area. However, because of its relative thinness,

articular cartilage transmits most of the load to the subchondral plate (77). In addition, the tangential collagen fibres at the surface of articular cartilage possess great tensile strength and are well designed to resist the shear forces of articular surfaces (78). The elastic nature of the cartilage matrix permits the ridges and mounds on the surface of articular cartilage to flatten as the surfaces glide together, further reducing friction.

2.3.2. Physical and mechanical properties of articular cartilage

Cartilage behaves as a viscoelastic material (79). The compressive modulus of articular cartilage is directly proportional to proteoglycan content (80). Kempson et al (1971) found that the stiffest cartilage had the highest concentration of glycosaminoglycans (chondroitin sulfate and keratan sulfate) (81). Proteoglycans hold water in the cartilage matrix osmotically and impede the loss of water from loaded cartilage both osmotically and by controlling matrix permeability. The high 'fixed charge density' due to the negatively charged groups on the proteoglycans leads to considerable 'swelling pressure' within cartilage (82). Collagen forms the structural framework of articular cartilage and resists shear and surface wear (tensile stiffness), compression loading with the help of

different orientation of collagen fibres at different regions. In addition, the collagen fibre network serves to counteract the swelling pressure elicited by the proteoglycans, contributing stiffness to the cartilage matrix (82). Disruption of the collagen network permits the tissue to swell. This may explain why fibrillated cartilage swells and contains more water (83) despite its lower proteoglycan content.

2.3.3. Morphology of normal, aging, degenerate lumbar facet cartilage

2.3.3.1. Normal cartilage

As a hyaline cartilage, the gross appearance of normal facet joint cartilage resembles those other articular cartilage. The cartilage surface is smooth, glistening and greyish white in fresh autopsy specimens. The cartilage is thickest, about 2 mm, towards the centre of the joint (37). The margins of the cartilage often displays small indentations into which small fringes from the synovial membrane indicate themselves.

Histologically, 4 zones may be identified in the cartilage (37). The superficial zone consists of 3 to 4 layers of ovoid cells whose long axes are parallel to the cartilage surface. Deep to this zone is a transitional

zone which cartilage cells are arranged in small clusters of 3 to 4 cells. Next deeper is a radial zone which consists of clusters of 6 to 8 large cells whose long axes is perpendicular to the cartilage surface and constitutes most of the cartilage thickness. The radial zone is only identifiable in the central regions of the cartilage. The deepest zone is the calcified zone which covers the subchondral bone plate, constitutes about 1/6 th of the total cartilage thickness.

2.3.3.2. Aging cartilage

It is not easy to differentiate the aging cartilage from early degenerate cartilage, both exhibit fibrillation changes. Taylor and Twomey had studied extensively for the age changes in facet joint cartilage (84,85,86). They found that the articular cartilage of facet joints steadily increases in thickness with age, but there may be some focal changes. Fibrillation (vertical tears and tangential splits) occurs more in the superior facet, predominately in the anteromedial, or backward-facing portion of this facet because this portion that resists the forward shear stresses applied to the intervertebral joint during flexion movement and weight-bearing. They observed that if severe and repeated stress were applied, then erosion and focal thinning of the cartilage would occur, while other region of the cartilage may exhibit swelling that accounts for the

general increase in thickness of the articular cartilage. In addition, they also demonstrated that the subchondral bone increases in thickness during growth and reaches maximum between 20 to 50 years of age, later it gradually gets thinner. In ostoearthritis the histology of the destructive lesions in exposed cartilage is often similar to that of age-related form of cartilage fibrillation, although differences are sometimes apparent in the advanced stage of the disease. Despite this histological similarity, the cartilage destruction seen in ostoearthritis of the hip may not simply be the result of severe age change, since on the femoral head the topographical distribution of age-related fibrillation and of the lesions of osteoarthritis appear to differ (87). However, on other synovial joints, it is possible that some instance of 'primary' osteoarthritis may develop from the type of cartilage fibrillation associated with aging: the patellofemoral joint of elderly women (88,89). Thus, it may be that, although the first naked-eye change in 'primary' osteoarthritis is always fibrillation, it does not always lead to osteoarthritis, being often a pathologically and clinically benign accompaniment of aging.

2.3.3.3. Degeneration in cartilage

Grossly, the initial appearance has a yellow discolouration and rough surface. Then extensive cartilage defects and osteophytes appear and occasionally fibrous and/ or true ankylosis would occur. Fibrillation and cell proliferation have been noted in the articular cartilage in early stages of osteoarthritis when it presents gross yellow disclouration and roughening of the surface. The cartilage changes would progress towards the rim of the cartilage with consequent loss of cartilage, and exposure of the subchondral bone plate (90). The above pathologies seem to be similar with other articular cartilage (91,92). However, both gross and microscopic examination of the articular surfaces in the same joint sometimes reveals considerable differences. The surface of one cartilage may have a normal cell pattern while the other surface may present with marked cell proliferation. Eisenstein (73) studied the pathological changes in the articular cartilage of the lumbar facet joints in patients with characteristic facet arthrosis syndrome. 12 patients with significant disability but normal plain radiographs were treated with local spinal fusion. The excised facet joint surfaces showed some of the histological changes seen in chondromalacia patellae and in osteoarthritis of other large joints.

2.3.4. The frequency and distribution of osteo-arthritis in lumbar facet joint cartilage

Guntz studied the thoracic and lumbar synovial joints in 56 autopsy cases. All the joints were opened and grossly examined for the presence of osteoarthritic changes (including the cartilage, capsule, any osteophyte formation and so on), which were graded according to their degree of degeneration. Some of the joints was also checked by microscopic examination. He found that below age 30 all joints were grossly normal. He also stated that the frequency of osteoarthritic changes are related to rising age, but he did not mention whether his figures were based on complete spines or the total number of joints examined. Degenerative changes were more frequent in thoracic regions than lumbar regions, especially at T3 to T6 levels. While in lumbar regions, L4-5 and L5-S1 were usually affected. A difference between the right and left sides was found only at the L1-2 and L5-S1 levels, the right side being predominant. Putti and Logroscino studied 42 male and 33 female lumbar spines from autopsy cases. The 4 spines in the 18-30 years age group all had grossly normal joints. All lumbar spines over 30 years of age manifested gross osteoarthritic changes which became progressively more severe with advancing age. Within each

spinal level, L1-2 showed the lowest frequency while the highest frequency was realized at L4-5 level. Ingelmark (93) studied spinal columns from a series of 138 male and 73 female skeletons. The criteria of osteoarthritis were: porosity of the articular surface, sclerosis of the subchondral bone plate, osteophytes in the joint capsule attachment region on the articular process, and ankylosis of the joint. These types of osteoarthritic changes showed a considerably higher frequency within the cervical and thoracic regions than the lumbar spine. No definite difference between levels could be demonstrated in the lumbar region, but L4-5 level showed the highest relative frequency. No differences were noted between sexes. Lewin (37) studied 86 adult lumbar spine from autopsy. He showed that there was no significant sex difference within any age group between any facets, any joints or any segments. The mean degree of osteoarthritic changes after age 65 significantly exceeded that in any age group up to 45 years, as did that in the 46-55 years group compared with the 20-25 years age group. Using the gross and microscopic as the assessment criteria for the degree of osteoarthritic changes, it showed that the degree of degeneration of lower lumbar segments (L3-4, L4-5, L5-S1, especially L4-5) were significantly higher than the upper lumbar segments. No significant differences were noted for the

superior or inferior facet, the left or right joint in the same segment or any of the lumbar segments.

2.3.5. Biochemistry of normal, aging and degenerate articular cartilage

Perhaps the morphology of normal or degenerate facet joint cartilage resembles other hyaline cartilage, therefore no substantial study investigates the biochemical changes of normal or degenerate facet joint cartilage. In the present investigation, the biochemical changes of the lumbar facet joint cartilage were referred to the background of our present knowledge about the biochemical picture in normal and osteoarthritis of other articular joint (knee, hip).

2.3.5.1. Normal cartilage

Normal human articular cartilage is composed of about 70 to 80 percent of water and equal parts of collagen and proteoglycans for the remaining constituents (20). Proteoglycan subunit has a linear protein 'backbone' to which are attached, 50 or more long side-chains of polydimeric sugars (glycosaminoglycans). At least three distinct species are identified: chondroitin 6-sulfate, chondroitin 4-sulfate and keratan sulfate. Both the water content and concentrations of

various glycosaminoglycans show variations within articular cartilage. Water content reaches its maximum in the middle zone of articular cartilage (94). The concentration of chondroitin sulfate and keratan sulfate rapidly rise as one progresses from the surface to one third of the depth of the cartilage and then it gradually diminishes (95). The absence of proteoglycans at the surface of the articular cartilage might be due in part to suppression of proteoglycan synthesis by synovial hyaluronate (96). The content and ratios of chondroitin sulfate are vary topographically over the articular cartilage of a joint (97) and also between different joints in the same animal (98). However, the greatest differences in chemical composition of cartilage are found between different individuals (99). The physiologic significance of these variations in water or glycosaminoglycan content remains unclear.

2.3.5.2. Aging cartilage

Little change occurs in the composition of normal human articular cartilage after the animal becomes skeletally mature. No appreciable change with age occurs in the water, collagen, total hexosamine, total chondroitin sulfate, total nitrogen, sulfur and ash content of the tissue (20). However, there is a shift from predominance of chondroitin 4-sulfate in young

people to chondroitin 6-sulfate and keratan sulfate in old age group (100). This may have some significance later in regard to the production of synovitis in osteoarthritis by polysaccharides (101).

2.3.5.3. Degeneration in cartilage

The proteoglycans of osteoarthritic cartilage is reduced, and the decrease is proportional to the severity of the disease (22). Although the total glycosaminoglycans is decreased, the individual species of the macromolecules are affected differently: keratan sulfate is relatively decreased and chondroitin 4-sulfate is increased, as compared to the normal state (100). Moreover, smaller and shorter chondroitin sulfate chains were demonstrated in osteoarthritic specimens (102). The above occurrence may represent changes in the production of link protein or increased degradation of glycosaminoglycan or the synthesis of immature forms (102,103). On the other hand, osteoarthritic human articular cartilage has a considerable increase in synthetic activity (20,22,104) and the rate of synthesis of proteoglycans being directly proportional to the severity of the disease process (105). But as the disease worsens, a point is reached at which the rate of proteoglycan synthesis falls off markedly, indicating

that the capacity of the cell to respond has been exceeded and the reparative function fails. The water content of osteoarthritic cartilage is significantly increased (106). This phenomena can be explained by a partial damage to the collagen network, which, as a result of this damage, is no longer able to restrain the swelling pressure of the proteoglycans (82). No substantial study investigates whether the increase in water content of articular cartilage is proportional to the severity of the disease process. The collagen content is unchanged (105) in osteoarthritic cartilage.

CHAPTER 3 - MATERIALS AND METHODS

3.1 Clinical study

3.1.1. Patients selection

Criteria for admission to the study:

1. Patients who were treated outside by general practitioner or referred from Accident & Emergency Department. They received physical therapy or antiinflammatory drugs outside for a period of time but no improvement, and referred them to special back clinics in Orthopaedics Department, Prince of Wales Hospital.

2. More than sixteen years of age.
3. Patients who fulfilled the diagnostic criteria of :
    a. Low back pain with or without radiation to the thigh, buttock or groin but not beyond the knee joint level.
    b. Paravertebral tenderness.
    c. Muscle spasm or spinal deformity.
    d. Painful motion (extension, lateral flexion).
    e. Sitting pain (static posture pain, which is improved by walking).
    f. Negative neurological examination.

65 patients were admitted to the study. Patients were reassessed during their time of follow up by the physiotherapists following the initial assessment.

3.1.2. Initial assessment

Standardized assessment forms(see Appendix 2) for the age, sex, occupation, duration of pain, the intensity of pain, functional limitation, and objective clinical findings were utilized by the physiotherapists when examining each patient. After being assessed, the 65 patients were allocated randomly into two groups of manual therapy and ultrasound therapy. The orthopaedic surgeon was in no way involved in any of the treatments given to the patients.

3.1.3. Reassessment

After the initial examination the patients were reassessed after three weeks, six weeks, nine weeks, twelve weeks. On these occasions the same assessment forms were used as when examining the patients initially. This standardized procedure for interviewing the patients ensured that each patient received an identical examination. The patient would be discharged if there was improvement and patient was satisfied after treatment, or occasionally their symptoms became worse then further investigation would follow.

3.1.4. Interpretation of the forms for the registration of related symptoms and signs

a. Pain intensity (both back pain and sciatic pain):

The patients were asked to choose a number ranged from 0 to 10 of their intensity of pain.

0: insignificant pain

10: terrible pain that they were not able to tolerate

1-9: moderate pain

b. Sitting pain:

Denoted the duration(in minute) before they complained of any low-back pain after sitting. If the patient complained that there is pain once they sit down, then we would record zero minute as the duration before the onset of the pain.

c. Functional score:

The patients were asked about their physical ability and daily activity of their life recently. We would record 0 to 5 score according to the following criteria:

5: fully functional

4: mild functional impairment (able to work)

3: significant impairment (occasionally sick leave)

2: community confinement (unable to work)

1: home bound

0: bed-ridden

d. Local tenderness:

The area of tenderness was registered by physiotherapist after thorough examination of the spine.

e. Range of motion:

Positive mark would be registered if there was pain denoted by the patient in the forward flexion, lateral flexion and extension movement. Negative mark for the absence of any pain.

3.1.5. Specific scoring system

1. Aims:

The "severity score" would be calculated and as an indicator of the general condition of the patients in each follow-up.

2. Interpretation:

The "severity score" ranged from 0 to 100. The higher the score, the higher the severity of the condition. The lower the score, the better the

condition.

3. Selection of the symptoms and signs:

The backpain, sitting pain, functional score, local tenderness and movement pain were selected as the basis to calculate the score by the conventional criteria (58,59).

4. Formula (see Table 2.)

Description		Formula	Minimum	Maximun
1.Backpain	Backpain score(B)	B x 2=___	0	20
2.Sitting pain	Duration in minutes(S)	(120-S)/6=___	0	20
3.Functional score	Score(F)	(5-F) x 2=___	0	10
4.Local tenderness	No. of tender points	One tender point: 10 Two or more points: 20	0	20
5.Range of movement	a.Lateral flexion pain (Left or right)	Positive: 10 Negative: 0	0	10
	b.Extension pain	Positive: 20 Negative: 0	0	20

Table 2. Formula for the calculation of 'severity score'.

3.1.6. Methods of treatment

1. Manual therapy

In this trial patients were allocated at random and treated by registered physiotherapists specially trained in manual therapy. Each treatment lasts about 15 minutes. The interval between manipulations is variable and is determined by the therapist (two or three manipulations per week is the most commonly used routine).

2. Ultrasound therapy

Patients in this study were randomly selected into this group. The interval between each treatment is also two or three times per week. Each treatment lasts about 5 to 10 minutes.

3.1.7. Statistical methods

Since the severity score is ordinal data rather than interval data, that is, score 50 is not equal to 2 times score 25. However, data distribution of the severity score is nearly in normal situation. Therefore, we can still use "multiple regression analysis" method to test which of the following variables were useful in predicting the severity score from patients who followed up to a specific period (after 3, 6, 9 weeks). Those were not followed up to the specific period would be excluded

from the analysis. Because only 5 patients followed up until 12 weeks, so no analysis had been done for this period.

1. Sex
2. Age
3. Occupation: housewife, sedentary, manual, others.
4. Acute or chronic duration: we classified the duration of symptoms less than or equal to 12 weeks as acute; those more than 12 weeks as chronic cases.
5. Type of treatment given: mainly ultrasound and manual therapy two groups of treatments.
6. Sessions of physiotherapy treatment:
   9 sessions (after 3 weeks treatment),
   18 sessions (after 6 weeks treatment),
   27 sessions (after 9 weeks treatment).
7. Initial severity score
8. $(Age)^2$*
9. $(Weeks)^2$*
10. $(Initial\ severity\ score)^2$*

For the convenient of analysis and let the frequency of the samples scatter more evenly, the "severity score" was stratified into 10 groups respectively, rather than using their original value.

* Remark: Square of the data may correct the abnormal distribution of the data in the analysis.

3.2. Cadaveric study of human lumbar facet joint cartilage

3.2.1. Cadavers

This investigation was based on observation made in a series of 24 lumbar spines from 24 subjects over 20 years of age. The five levels of lumbar facet joints, both left and right, totally 240 joints were cut out as blocks at autopsy from subjects with no evidence of traumatic joint disease, malignancy , rheumatoid arthritis or tuberculosis.

The age and sex distribution of the 24 adult lumbar spines was as follows:

age group	Men	Women	No.
20-40	3	4	7
41-60	4	0	4
>61	8	5	13
Total no.	15	9	24

The series of lumbar spines was unselected in the sense that they did not come exclusively from subjects with or without back disorders during life.

3.2.2. Procedures of dissection of the lumbar facet joint and articular cartilage

After all the spinal muscles were stripped away, T12 vertebra was identified (T12 was defined as the lowermost vertebra bearing a rib). Then the five levels of facet joints below T12 vertebra, both left and right sides, were cut out as blocks with the help of osteotome and other equipments. The joints were examined and further procedures were carried out as soon as possible. Cartilage from different degree of degeneration from each facet joint were dissected with bone and other tissues carefully excluded. Each tissues from different degree of degeneration of each cadaver were pooled in order to get sufficient samples for further investigation.Samples were stored at $-20^{o}C$.

3.2.3. Development of "indices" for the observation and statistics of the articular cartilage in facet joints

In order to describe the degenerative pattern of the articular cartilage, and delineate the relationship between gross morphology and histomorphology in normal or

degenerated articular cartilage. Three types of "indices" were introduced.

3.2.3.1. "Gross Morphological Index"

Based on the gross appearance of articular cartilage in knee or hip joint, that is, their colour, brightness and the smoothness of the cartilage. Bennett (107) and Collins (108) classified them into 4 different grades. But now we are using 3 different grades because grade 2 and grade 3 is fairly difficulty to differentiate. The criteria were as follows:

Grade I(normal): smooth, white(sometimes pale yellow), glistening, firm.

Grade II(moderate degeneration): softening, fraying

or rough surface. Thinning of the cartilage. Also may have erosion, ulceration but not exposing the subchondral bone.

Grade III(advanced degeneration): marked erosion of the surface, only a thin layer of soft, irregular cartilage. Complete erosion and exposure of subchondral bone may occur.

Procedures:

After opening the facet joint, the superior(concave) and inferior(convex) facet total 460 articular surfaces

were examined separately. The normal and abnormal patterns of each surface was recorded down onto the paper. The edges and the proportion of each grade would be taken into consideration when drawing. The exact proportion or percentage of each grading was obtained after cutting, weighing each portion of grading using a electrical balance (109). The proportion or percentage of each grading were then multiply their original grading. Furthermore, "Gross Morphological Index"(GMI) was obtained after adding the numbers together. For example, {(20% x 1[Grade I]) + (60% x 2[Grade II]) + (20% x 3[Grade III])} =2. Finally, each surface of facet joint would have a "GMI" to denote their severity of degeneration of the articular surface.

Figure 1 is an example for the determination of 'Gross Morphological Index'.

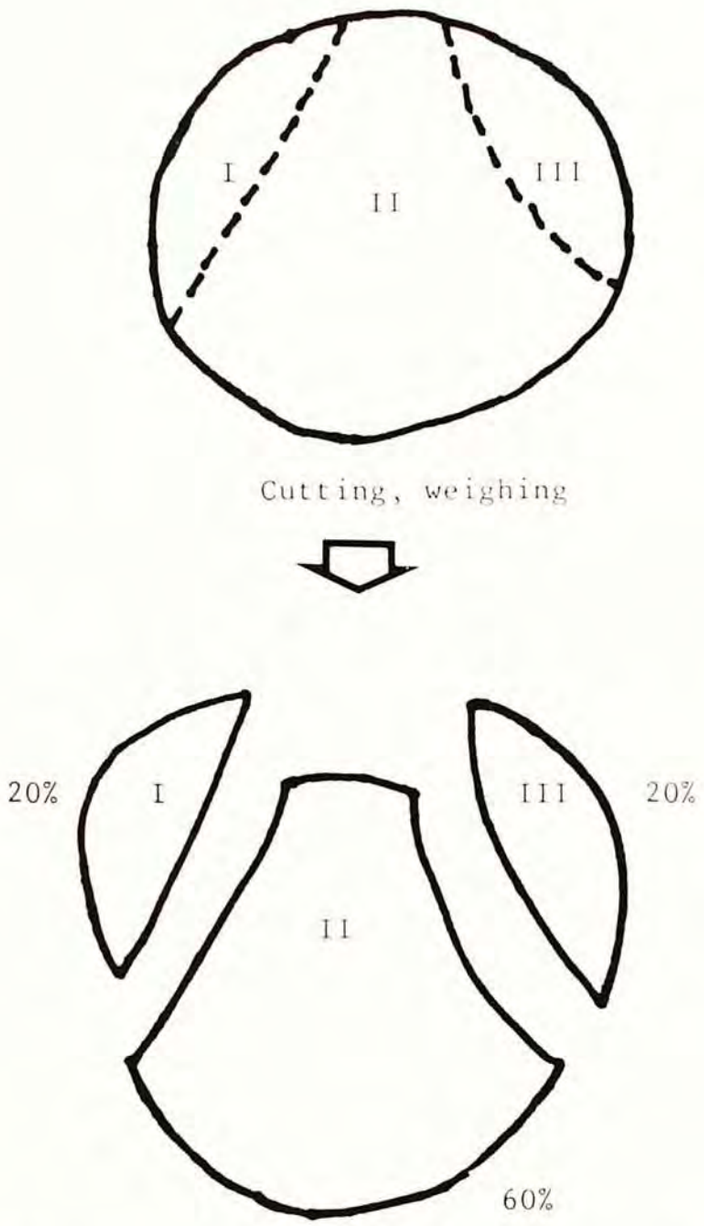


Figure 1. Example for the determination of 'Gross Morphological Index' (GMI). (% by weight)
'I' denotes normal cartilage, 'II' denotes moderate degenerative cartilage, 'III' denotes advanced degeneration cartilage.

'GMI'= (20%x 1(I)) + (60%x 2(II)) + (20%x 3(III))
= 2.0

3.2.3.2. "Gross Linear Index"

In order to prove the gross observation is the true representatives of the normal or abnormal patterns of the articular cartilage. Histological sections had been done for the 40 random selected articular surfaces. Each selected articular surface was cut using diamond saw and the cutting line was marked onto the previous paper for recording the "GMI" on the articular surfaces. The proportion or percentage of each grade upon the cutting line were obtained by measuring the linear distribution of each grade on a ruler. 'Gross Linear Index' was calculated using the previous formula.

Figure 2 is an example for the determination of "Gross Linear Index".

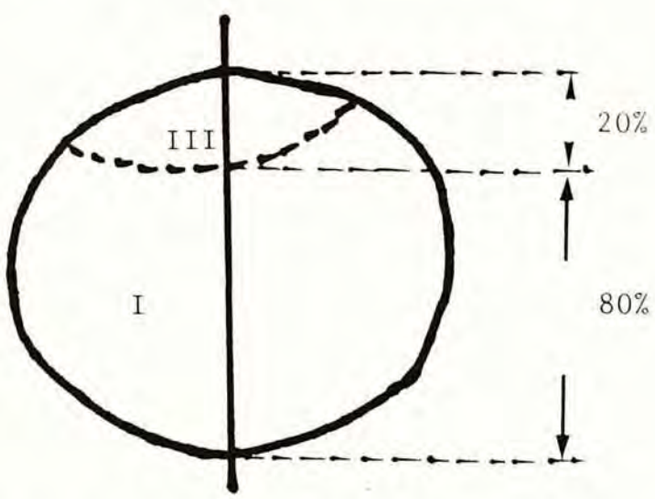


Figure 2. Example for the determination of ' Gross Linear Index' (GLI).(% by length)
'I' denotes normal cartilage, 'III' denotes advanced degeneration cartilage.
'GLI'= (20%x 3(III)) + (80%x 1(I))
= 1.4

3.2.3.3. "Histological Linear Index"

Those sectioned samples were followed by standard paraffin section procedures. Then haematoxylin & eosin staining was performed. We used the modified grading criteria by Weiss (110).

Grade I: Intact surface, no fibrillation, 4 zones (superficial, transitional, radial, calcified) can be identified.

Grade II: Fibrillation, either vertical or horizontal splitting of the articular surface. Cluster of chondrocytes formation. Destruction of tidemark.

Grade III: Full thickness of cartilage loss or disorganization. Subchondral bone exposure and there may have fibrous or fibro-cartilage recovery of exposed bone.

Procedures:

Each sectioned slide was examned randomly in order to reduce the bias from the examiner. The proportion or percentage of each grade on each surface were obtained by measuring the linear distribution of each grade under the microscope. "Histological Linear Index" was calculated using the previous formula.

Figure 3 is an example for the determination of "Histological Linear Index".

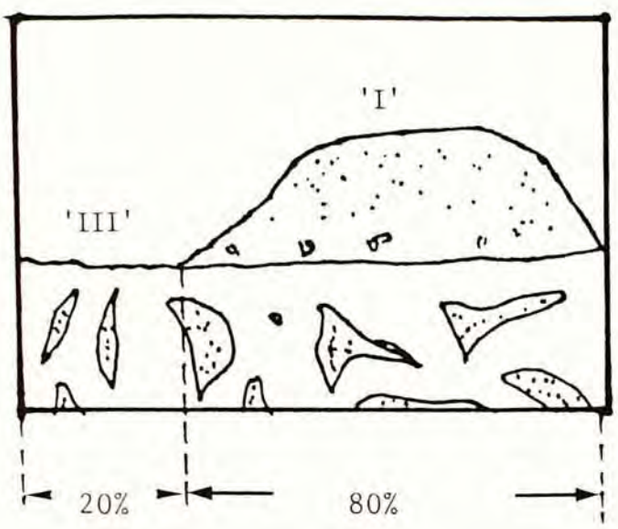


Figure 3. Example for the determination of 'Histological Linear Index' (HLI) under microscope. (% by length) 'I' denotes normal cartilage, 'III' denotes advanced degeneration cartilage.

'HLI'= (20%x 3(III)) + (80%x 1(I))

=1.4

3.2.4. Biochemical study of normal and degenerate cartilage

3.2.4.1. Water content

A modified method from Simon (111) was used for the determination of water content of the cartilage. Slices of cartilage from each grade were blotted dry by gauze to remove excess water from the surface of the cartilage.

Cartilages were diced into about $1mm^3$ size inside a humidified box in order to prevent water loss by evaporation. Randomly selected samples weighed at least 50 milligrams by electrical balance from each grade were put into the Eppendorf microcentrifuge tube (112), weighed, then wet weight was obtained. Samples were lyophilized (113) for about 16 hours. Finally, re-weighed the samples to get the dry weight. The dry weight divided by the wet weight provided a percentage value for the solids, which when subtracted from 100 gave an estimate of the percentage of water content in the cartilaginous tissues.

3.2.4.2. Proteoglycan content

The detection of uronic acid was carried out by the modified method of Bitter and Muir (114). In which, standard chondroitin 6-sulfate (115) is used as the standard reagent rather than glucuronolactone.

We used the modified method of Bollet (116) for the extraction of uronic acid component from the cartilage. However, standard chondroitin 6-sulfate was used for the comparison between this modified method and other enzymatic method for the extraction of uronic acid (106), in order to show the consistency of uronic acid yield by these two methods. The procedures and results were shown in Appendix 3.

In addition, experiments had performed to test the stability of standard chondroitin 6-sulfate in the condition of this modified method and other conditions, that is, in deionized water or 6 mol/l sulfuric acid with and without heating at 90^{o}C for 45 minutes. The procedures and results were shown in Appendix 4.

Reagents:

(a). Sulfuric acid (6 mol/l)

Prepared from 95-97% sulfuric acid, specific gravity= 1.84 (analytical grade). 32 ml 95-97% sulfuric acid was added to 68 ml deionized distilled water in a cool environment.

(b). Sodium tetraborate (117) (0.025 mol/l)

In sulfuric acid, specific gravity= 1.84 (analytical grade). 0.9535 g sodium tetraborate was dissolved in 100 ml sulfuric acid.

(c). Carbazole (117) (0.125%)

In absolute ethanol (analytical grade). 0.0125 g carbazole was dissolved in 10 ml absolute ethanol.

(d). Chondroitin 6-sulfate standard (5-160 ug/ml)

Prepared by dilution with 6 mol/l sulfuric acid.

Procedures:

(1). Extraction:

Pooled and diced cartilage from each grade weighed 10 to 15 milligram and was put into glass tube. It was heated in water bath at about 90°C for 45 minutes. The tubes were cooled and centrifuged at 3500 rpm for 15 minutes in room temperature. The supernatant was diluted to 1:5 before detection.

(2). Detection:

1.5 ml sodium tetraborate reagent was placed in tubes with Telfon stoppers and cooled to 4°C. 0.25 ml sample or standard was carefully layered onto the acid. The tubes were closed and shaken at first gently, then vigorously with constant cooling. The tubes were then heated for 10 minutes in a vigorously boiling distilled water bath and cooled to room temperature. Carbazole reagent (0.05 ml) was then added, the tubes were shaken again, heating in boiling bath for a further 15 minutes. and cooled to room temperature. The absorbance (A) was then read at 530 mu in a 1 cm cell using a spectrophotometer (118).

3.2.5. Statistical methods

3.2.5.1. Correlation between "Gross Linear Index" and "Histological Linear Index":

Since these "Indices" are ordinal data rather than interval data, that is, "Index" grade II is not equal to grade I times 2. Therefore, "Spearman rank correlation coefficient" is used to analyse the correlation between these ordinal measures. No equation or fit line will be obtained for this type of analysis.

3.2.5.2. Correlation between "Gross Morphological Index" and other related factors

The "Gross Morphological Index" (GMI) will be tested for the correlation with the age, sex, facet joint levels, left or right side, and superior or inferior facet, $(age)^2$. The 'Multinomial Logit Regression' is used for analysis the correlation between the ordinal data 'GMI' and other factors. The original 'GMI' was stratified into 3 categories for the ease of analysis, that is, grade I= (>=1 to <1.666), grade II= (>=1.666 to <2.333), grade III= (>=2.333 to <3). Regression analysis will result two equations with those correlated factors to obtain a certain value. These values are used for calculation of the probability for "predicted" gradings I, II and III. The highest probability value among these three "predicted" gradings will be chosen as the final "pre-

"predicted" grading. The original 3 categories of grading will be compared with the "predicted" grading. There is such a correlation if the three "predicted" gradings classified correctly 70% of the gradings into these original 3 categories.

3.2.5.3. Correlation between "Gross Morphological Index" and water, proteoglycan content of the cartilage

Student t-test of multiple comparison is used for testing the correlation between grade I(normal), grade II(moderate degeneration), grade III(advanced degeneration) facet joint cartilage.

CHAPTER 4 - RESULTS

4.1. Clinical study

4.1.1. General results of patients

4.1.1.1. Sex and age

The distribution by age and sex in the studied population is illustrated in Figure 4. About 80% of the patients in the study were women. The youngest patient in this study was 16 years of age and the oldest was 68 years of age. The mean age was 33.7 years.

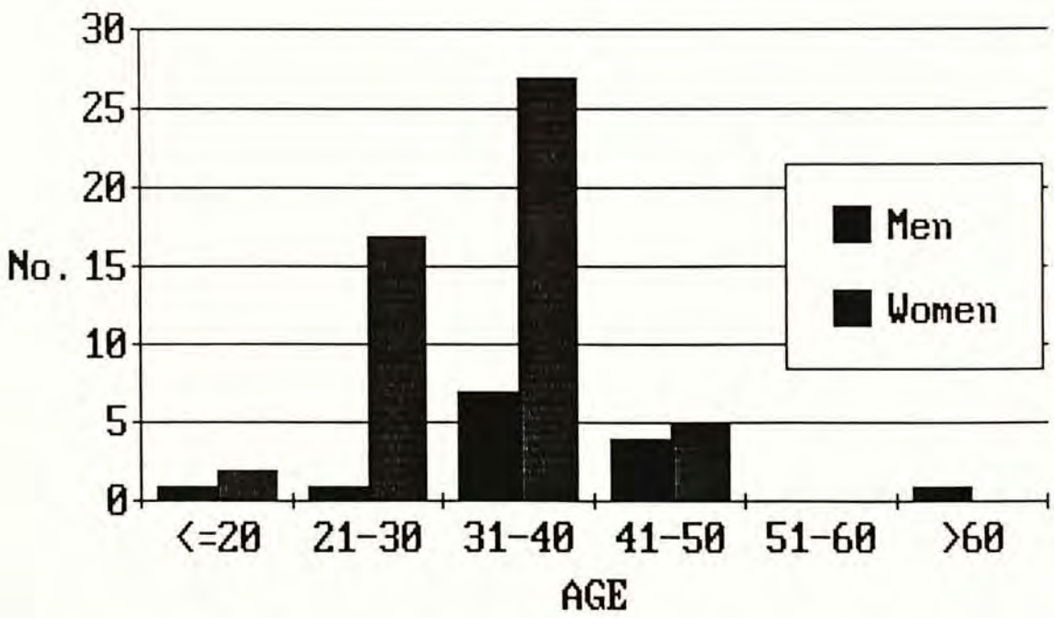


Figure 4: Distribution of age and sex in patients with facet syndrome

4.1.1.2. Occupation

Most of the patients were housewives (42%) and sedentary workers (25%), only few of them were manual workers (5%), as illustrated in Figure 5.

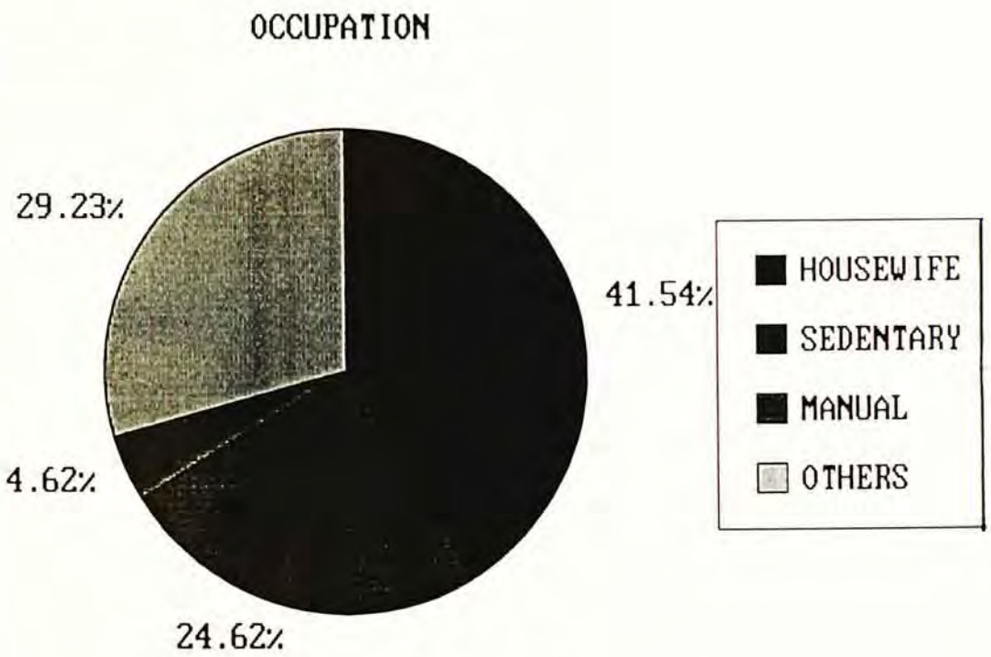


Figure 5: Occupation in patients with facet syndrome

4.1.1.3. Duration of symptoms before entry to the study

The time between the onset of symptoms and the initial assessment of the patient ranged from 1 week to 20 years. Most patients (83%) had their back pain for more than 12 weeks at the first examination. The median duration of symptoms was about 1 year. This is shown in Table 3.

Duration of symptoms (weeks)	Number of patients
less than or equal to 12 weeks	11
13-24	6
25-36	6
36-52	10
52-104	9
>104	23

Table 3. Duration of symptoms before entry to the study

4.1.1.4. Subjective symptoms

All patients complained of low-back pain, about half of them considered the pain to be intensive (score more than 5). Only about 30% of patients complained of sciatic pain and about 90% of them considered the pain as mild to moderate (score less than or equal to 5). 67% stated that they had sitting pain while 15% of them had impulse pain (pain was accentuated by coughing and sneezing). The different varieties of pain in the group of patients are presented in Table 4.

	Number of patients			
	Yes	No	Intense pain (score >5)	Mild to moderate pain (score <=5)
Backpain	65	0	31	34
Sciatic pain	18	47	2	16
Pain on sitting	44	21	-	-
Impulse pain	10	55	-	-

Table 4. Frequency of different varieties of pain.

The degree of functional limitation due to back pain is listed in Table 5

	Number of patients
5. Fully functional	0
4. Mild functional impairment (able to work)	5
3. Significant functional impairment (occasionally sick leave)	56
2. Community confinement (unable to work)	3
1. Home bound	1
0. Bed-ridden	0

Table 5. Functional limitation of activities of daily living.

4.1.1.5. Objective findings

An increase of pain when extension was reported by 95% of the patients while increase of pain was not so frequent in the other plains of motion. The Straight Leg Raising test (SLR) was performed and only 5% of the patients had an SLR below 60°. Only few patients were found to have neurological disturbance. These clinical findings are presented in Table 6.

	No. of patients	
Clinical findings	Yes	No
Increase pain on: extension	63	2
right lateral flexion	33	32
left lateral flexion	26	39
forward flexion	37	28
Neurological disturbance: motor	3	62
sensory	2	63
jerk	2	63
SLR <60°: left side	3	62
right side	3	62

Table 6. Objective findings.

Tenderness was registered mainly located centrally in the lower lumbar area or paravertebral regions as illustrated in Table 7.

Tender points		No. of patients
Central:	Thoracic area	1
	L1-2 level	3
	L2-3 level	7
	L3-4 level	16
	L4-5 level	38
	L5-S1 level	45
Paravertebral: left	L1-2	1
	L2-3	8
	L3-4	11
	L4-5	12
	L5-S1	14
	right L1-2	1
	L2-3	6
	L3-4	9
	L4-5	8
	L5-S1	14
Sacral area:		7
Posterior sacro-iliac joints:	left	8
	right	7

Table 7: Distribution of tender area on the lumbar regions.

4.1.2. Influence of various variables in predicting the "severity score"

After calculation, different patients will have different initial scores. The severity scores, in general, decreased with weeks of physiotherapy treatment. However, after the statistical calculation by multiple regression, the adjustment of the "score" will result a "predicted severity score", which in turn indicate the quantitative value in correlation with those depending variables. The following table (Table 8a, 8b) illustrates the general profile of the patients in different follow-up period, and indicates which factors have highly correlation with the "Predicted Severity Score" (P.S.). The equations for calculation the "P.S." and their multiple correlation coefficient are shown also. From the equation, we can find that "P.S." is decreasing significantly as increasing weeks. The decrease of "P.S." in acute cases is more pronounced than those chronic cases in any period. However, the "P.S." of chronic cases is always higher than those acute cases. The decreased amplitude of cases having either higher or lower initial score are equal.

	Follow up until 3 weeks	Follow up until 6 weeks	Follow up until 9 weeks	FU till 12 weeks
Sample size	65	43	17	5
Sex:				
Men	14	8	5	0
Women	51	35	12	5
Age:				
Range	16-68	16-46	16-46	26-33
Mean (SD)	33.7 (7.8)	33.3 (6.2)	33.8 (7.8)	31 (3.1)
Initial score:				
Range	3-10	3-10	6-10	7-8
Mean (SD)	7.2 (1.2)	7.3 (1.3)	7.9 (1.1)	7.8 (0.8)
Occupation:				
Housewife	27	19	7	3
Sedentary	16	10	2	1
Manual	3	3	1	0
Others	19	11	7	1
Duration:				
Acute (<=12 weeks)	11	6	2	0
Chronic (>12 weeks)	54	37	15	5
Treatment:				
Manual therapy	38	22	8	4
Ultrasound therapy	27	21	9	1
Correlated factors:				
High correlation:	1. Initial Score (I.S.)	Same as left	Same as left	No analysis
	2. Weeks of treatment (Wk)			
	3. Duration of symptoms: acute or chronic			
No correlation:	1. Types of treatment	Same as left	Same as left	No analysis
	2. Sex			
	3. Age			
	4. Occupation			

Table 8a. General profiles and results of multiple regression on different period of follow-up.

	Equations:	Equations:	Multiple correlation coefficient	
	Acute	Chronic	Acute	Chronic
FU until 3 weeks	"P.S."=(0.9854xIS)-(0.8434xWk)	"P.S."=(0.9958xIS)-(0.4653xWk)	0.77	0.75
FU until 6 weeks	"P.S."=(0.9882xIS)-(0.5593xWk)	"P.S."=(1.0002xIS)-(0.3265xWk)	0.80	0.78
FU until 9 weeks	"P.S."=(1.0033xIS)-(0.6473xWk)	"P.S."=(0.9939xIS)-(0.2267xWk)	0.88	0.61
FU until 12 weeks	Nil	Nil	Nil	Nil

Tables 8b. Equations for the calculation of "P.S." and their related multiple coefficients.

4.2. Cadaveric study of human lumbar facet joint cartilage

4.2.1. Sex and age

Total 24 cadavers, with 15 men and 9 women were studied. The average age is 61, with a range from 20 to 87 years. The distribution of age and sex is shown in Figure 6.

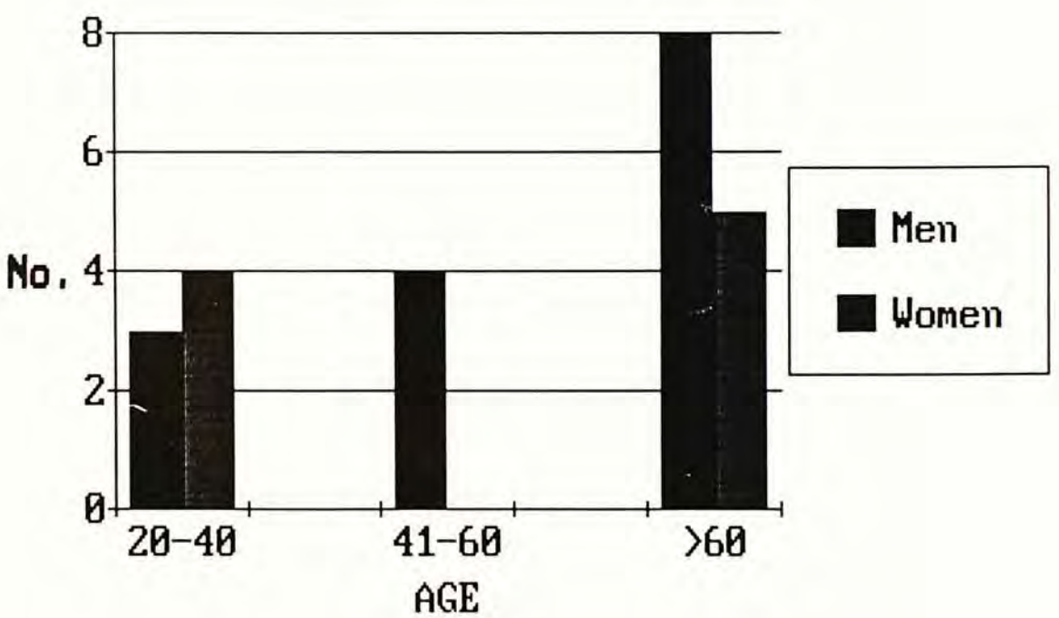


Figure 6: Age and sex distribution of cadavers

4.2.2. The correlation between gross and histological appearance

There were 40 randomly selected articular surfaces for histologic sections. The result shows that there is a high correlation between the naked eye and microscopic classification of the articular cartilage. The coefficient of correlation is 0.74 with P value < 0.001. The correlation is illustrated in Figure 7. Examples for gross and histological appearance are shown in Figure 8 to Figure 16.

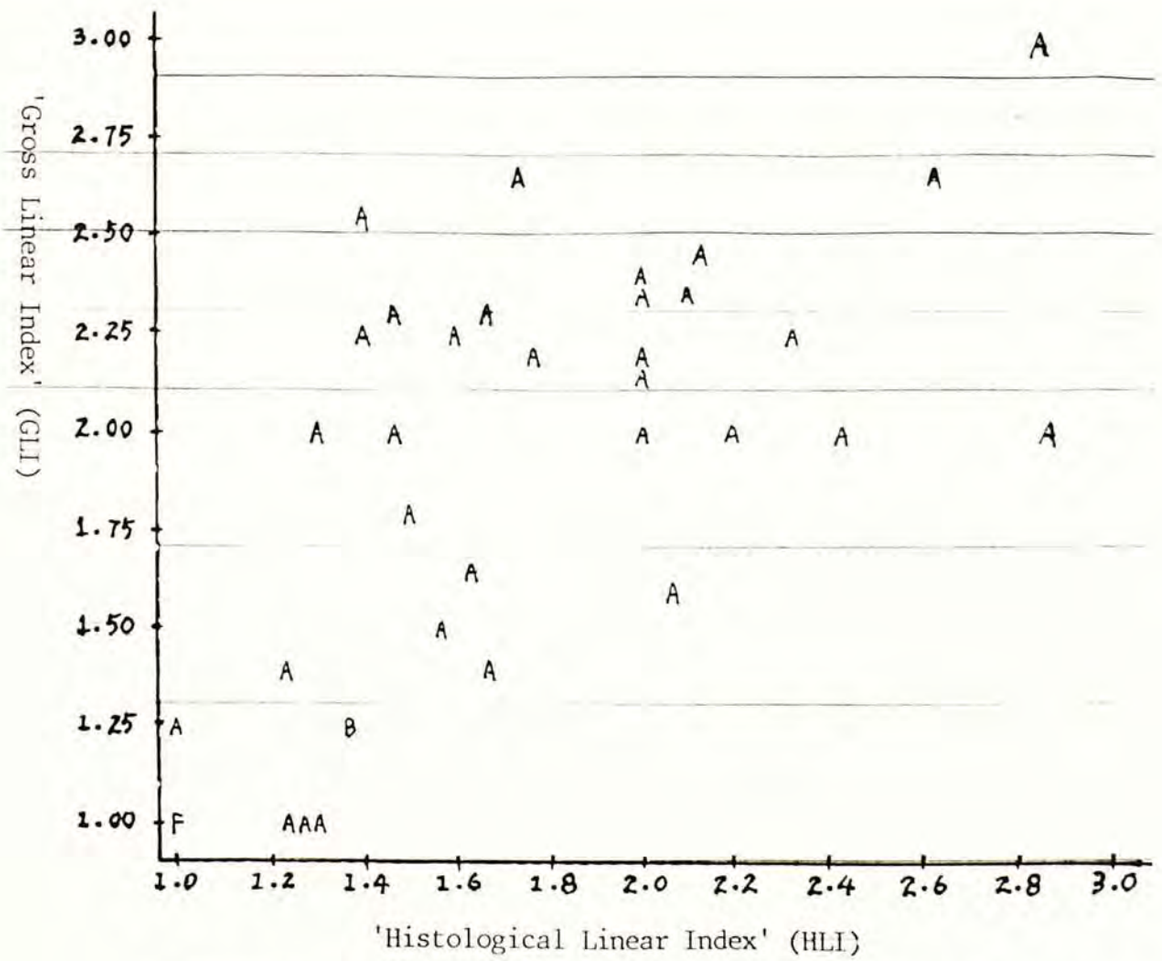


Figure 7. Correlation between Gross and Histological "Indices".

4.2.3. Osteoarthritic changes in the lumbar facet joint cartilage related to other factors.

The total 460* "Gross Morphological Index" (GMI) had been used to test for the correlation with the age, sex, lumbar level, left or right side, superior or inferior facet, and $(age)^2$. General frequency distribution of various related factors is shown in Table 9.

After calculation, the other 4 levels (L12, L23, L34, L5S1) were quite different from level L45, therefore the 4 levels were classified into a separate group as to compare with the level L45 group. The results show that there is a significant correlation between the "GMI" and age, sex, level of lumbar spine, $(age)^2$. No correlation is shown between "GMI" and the left or right side, superior or inferior facet. However, the "predicted" grading shows less than 70% comparing with the relative original grading (see Table 10). Furthermore, no "predicted" grading is found in grade III. Therefore, the original grading is not useful to determine the "predicted" grading although some factors have correlation with the "predicted" grading.

Nevertheless, the results still disclose that the "predicted" grading shows a tendency to increasing with advancing age. No obvious difference between male and female except for older female, the "predicted" grading seems to be higher than the other old male. The "predicted" grading of level L45 displays a better tendency when comparing with the other 4 levels.

* Remark: 20 "GMI" were discarded because these were classified into 4 gradings initially.

Factors	No. of articular surfaces
Grade:	
I ("GMI" >=1 to <1.66)	268
II ("GMI" >=1.66 to <2.33)	141
III ("GMI" >=2.33 to <3)	51
Sex:	
men	312
women	148
Side:	
left	230
right	230
Superior or inferior:	
superior	231
inferior	229
Levels:	
L12	93
L23	91
L34	92
L45	93
L5S1	90

Table 9.
Frequency distribution of various related factors with "GMI"

		Predicted grading			
		I	II	III	Total no.
Original grading	I	185 (69%)	83 (31%)	None	268
	II	98 (69.5%)	43 (30.5%)	None	141
	III	30 (58.8%)	21 (41.2%)	None	51
Total no.		313	147	0	460

Table 10.
Correlation between the original & "predicted" 3 categories of grading.

4.2.4. "GMI" and water content of the cartilage

"GMI" grade I, II, III, that represent normal cartilage, moderate degeneration, advanced degeneration. The water content of normal cartilage from 48 specimens averaged 67.8%, whereas in moderate degeneration from 51 specimens it averaged 71.4% of the wet weight. The P value is < 0.001. The water content of cartilage with advanced degeneration averaged 69%, compared with normal and moderate degeneration, P value is > 0.05. The results are shown as the following Table 11.

	No.	Water content(%)	SD
Normal	48	67.8	3.66
Moderate degeneration	51	71.4	3.08
Advanced degeneration	8	69	7.08

Table 11: The water content of normal and degenerate cartilage.

4.2.5. "GMI" and proteoglycan content of the cartilage

Since the proteoglycan content is reflected by the chondroitin sulfate content, hence the uronic acid of the cartilage. The uronic acid content of normal cartilage averaged 85.9 (μg/mg dry cartilage), which shows no significant difference (P > 0.05) when compared with moderate degeneration cartilage. However, there is a highly significant difference (P < 0.001) when compared the uronic acid content of advanced degeneration cartilage with the normal or moderate degeneration cartilage.

The results are illustrated in Table 12.

	No.	Uronic acid content(µg/mg dry)	SD
Normal	37	85.9	23.3
Moderate degeneration	33	78.2	19.7
Advanced degeneration	22	51.0	19.5

Table 12: The uronic acid content of normal and degenerate cartilage.

CHAPTER 5 - DISCUSSION

At any one level of the spine, the motion segment is constituted of three distinct parts: the two facet joints and intervertebral joints (the disc). Any trauma or pathological changes in one part of the complex will, in time, consequently spread to the two others through changes in the mechanical behavior of the construct. As the only synovial joint in the spine, when the movements between the spinal segments become uneven, excessive, or irregular, dysfunction in facet joints with following degenerative changes will appear.

The 3 stages theory proposed by Kirkaldy-Willis (52), for explaining the pathogenesis of degenerative changes in any synovial joints suggested that the facet pathology of the first phase (phase of dysfunction) will exhibit itself in the facet syndrome.

Since the diagnostic criteria for facet syndrome is still controversial, therefore the incidence or prevalence of this syndrome varies. A retrospective review of 1293 cases of low-back pain treated over a 12-year period by Benard and Kirkaldy-Willis (1987) (10), revealed that 22% patients experienced facet syndrome. Local experience revealed that there was about 12% of low-back pain out-patients diagnosed as facet syndrome according to the general accepted criteria (unpublished material).

In the clinical study, the average age of the patients was 33.7 years, younger age group compared with other studies for patients with facet syndrome (13,119). This phenomena may be due to relatively different criteria for selection of patients into the study. On the other hand, the three times more common in female is consistent with the other study (119). That may be

related to their greater lumbar lordosis, which means that each synovial joint is more extended than in the male. This may further lead to increase articular pressure and accelerate the degenerative process of facet joints. The predominance of housewives and sedentary workers may also be interpreted by rigours of home caring and prolonged sitting posture.

The overlooking of facet syndrome and follow by inappropriate treatment protocol may explain the chronicity of the present study. The other reasons may be due to the combined pathologies overshadowing the significance of facet joint pathology, and the lower incidence of lock facet syndrome in acute cases.

The higher incidence of back pain and sitting pain of present study indicates the origin of pain may come from facet joint pathology. While the indicators of radicular pain, sciatic pain and impulse pain, show a low incidence. The limited information obtains by asking one question to assess the degree of functional disability is still useful in certain aspect. Most patients experience significant functional impairment (occasionally sick leave), which might indicate the significant effect of facet joint in the contribution of functional daily activities. The more detailed question of functional limitation of low-back pain patients proposed by others (120), may reflect the true functional status more accurately although no substantial questions specific to assess the disability of facet syndrome is available. However, this type of subjective information can be influenced by errors characterizing either the investigator or the investigated individual themselves.

Most patients (95%) experienced pain on extension, this may be explained by the increase in the facet articular pressure during extension movement (49). About

half of the patients had increase pain on the other plane of motion of the spine. Only few patients had neurological disturbance and Straight Leg Raising test below 60^{o}, which were specific for the diagnosis of radicular origin. Tenderness was registered mainly centrally and paravertebral regions in the lower lumber area (L4-5, L5-S1) which agrees with the general distribution of facet syndrome by other authors (13,119).

The validity of diagnoses of "facet syndrome" may also be questioned in view of the lack of specific signs or symptoms, or investigation regarding the pathophysiology of facet joint disorders. In this study attempt was made to achieve homogeneity of diagnosis in the studied group by including patients with conventional signs or symptoms for facet syndrome.

The validity of the scoring system in the clinical study may be demonstrated by the significant decrease of scores with increasing time of physiotherapy treatment. The "Predicted Severity Score" had a highly correlation with the initial score, time of physiotherapy treatment, acute or chronic duration of symptoms, while the type of treatment (whether manual or ultrasound therapy), age, sex, occupation were not useful in predicting the "score". In addition, the results disclose that for chronic patients (duration more than 12 weeks), the "Predicted Score" is always higher than the acute patients (duration less than or equal to 12 weeks). However, with weeks of treatment, the decrease of "score" is more effective for acute cases. Most of those disability cases with acute onset would consult emergency department rather than going to the out-patient clinics. This may explain the reason for the lower "score" in acute cases in certain aspect. It seems difficult to explain the fact that the decrease of "score" was the

same for patients with higher or lower initial score, however, the effectiveness of the treatments may give some of the explanation. It is not possible for the present study to determine the appropriate duration of treatment for different patients due to the lack of information after the last follow-up period. Nevertheless, the decrease of 'score' is not related to type of treatment, which implicates the ultrasound treatment as effective as manual therapy. Since manual therapy is practiced only by specially trained physiotherapist, therefore in future, in order to be more economic in clinical practice, ultrasound treatment should be the first line treatment for patients with facet syndrome.

Low-back pain is often described as periodic in nature with spontaneous remissions. The natural history of low-back pain, that is, without any treatment given, is still favourable. Since the present study is prospective, we cannot compare the outcome of the treatment groups with the placebo treatment group or the non-treatment group. Most discharged patients showed significant improvement after treatment as assessing by the physiotherapists and surgeons, however, some patients became worse after treatment but still discharged for further management. Furthermore, treatment was not homogeneous, other modalities of treatments had been given during the course of treatment. The lack of information after the last follow-up makes it more difficult to determine the long-term effect of physiotherapy treatment, although most people believe that only short-term improvement will be the result of physiotherapy. As a result, in future study on the effect of physiotherapy for low-back pain patients, a strictly homogeneous treatment protocol should be established and long-term follow-up of all the patients should also be

proposed. Ideally, a separate group of patients without any treatment given, or with placebo treatment should be utilized to study the natural course of low-back pain after different modes of treatments.

The morphological (histological) changes of articular cartilage has been recognized as the earliest changes during the degeneration process of synovial joint. However, the relationship between these degenerative changes of facet joint cartilage and symptoms is still difficult to correlate. Eisenstein and Bough (73,74) had examined the facet cartilage in patients with typical facet syndrome presentation and under local spinal fusion operation. The results showed that the excised facet joint surfaces demonstrated some of the histological changes similar in chondromalacia patellae and in osteoarthritis of other large joints. However, in a postmortem study such as this, clinical correlation is not possible and further deduction about joint function are difficult by referring to the appearance of the cartilage only.

Most of the cadavers in the present study belong to the old age group, only few of them are younger age group due to the difficulty of getting the samples. The high correlation between the naked eye and the microscopic classification indicates the validity of naked eye observation of the articular surface. In future, in order to achieve more accurate classification for the degree of degeneration of facet joints, the other structures (subchondral bone, capsule, synovium, osteophyte) should also be considered. Furthermore, histochemical techniques for the articular cartilage is useful to delineate the biochemical distribution of various components inside the cartilage matrix.

The degree of ostoearthritic changes of facet cartilage, as reflected by "Gross Morphological Index", is significantly higher in more than 60 years age group

than the 41 to 60 years age group, also the 'GMI' is significantly higher in 41 to 60 years age group than the 20 to 40 years age group. This phenomena is consistent with other studies for facet joint (37) and other synovial joints. Aging is still an important factor in the pathogenesis of ostoearthritic of synovial joint, although the etiology of osteoarthritis is unclear. The higher degree of degeneration in older women than men may be due to the presence of various factors correlated with the pathogenesis of osteoarthritis. More sedentary workers and lordotic spine after delivery were found in women, which may have significant effect in the development of pressure related pathologies of facet joints. In addition, the weaker back muscle development in women may have influence for the normal stability of the spine. Therefore, in future study, Cybex machine may be utilized for the assessment of back muscle in the contribution of stability of the spine. Moreover, pressure transducer device may have significance for recording the articular pressure during each movement as to justify the pressure effect of sitting posture or extension on the articular surfaces. It is difficult to interpret that the level L45 showed a lower degree of degeneration as comparing with the other 4 levels. On the contrary, other study (37) demonstrated that the L45 level showed the greatest degree of degeneration among the different spinal levels. However, the poor correlation between the original grading and the "predicted" grading implies that there is some other factors, which are not included in the present study, may have some contribution to disclose their substantial relationship. Further study should collect more specimens and using a more detailed classification of the gross appearance of the cartilage may be employed to justify the above phenomena.

The lack of knowledge of biochemical composition in normal and degenerate facet joint cartilage makes the result difficult for comparison. Assuming that the cartilage of facet joint behaves in the same way as the other synovial joints, the significant increase in water content for the cartilage with moderate degeneration in the present study correlates well with other study done on the other articular cartilage. This can be explained by a partial damage to the collagen network, which, as a result of this damage, is no longer able to restrain the swelling pressure of the proteoglycans (82). The absence of significant increase in water content for the advanced state of degeneration as compared with normal and moderate state of degeneration, may be explained by the fact that only deeper layer of cartilage is left for the detection of water content, which contains less water than the middle zone (94). The small sample size and higher standard deviation value may have some influence for the insignificant difference. The significant decrease in proteoglycan (chondroitin sulfate) content for advanced stage of degeneration shows consistency with other studies (22) on other synovial joint cartilage. However, the proteoglycan content between moderate state and normal state of cartilage revealed no significant difference. The rate of synthesis of proteoglycans in osteoarthritic cartilage has increased to a certain extent, and the rate of synthesis of proteoglycan being proportional to the severity of the disease process. The decrease of proteoglycan and increase synthesis of proteoglycan may come to a balance. In that case, no significant decrease of proteoglycan is detected. Nevertheless, as the disease worsens, a point is reached at which the rate of proteoglycan synthesis falls off markedly, indicating that the capacity of the cell to respond has been exceeded and the reparative function fails. Concerning the different species of

glycosaminoglycans in degeneration for other articular cartilage, keratan sulfate is relatively decreased and chondroitin 4-sulfate is increased, as compared to the normal state. The present study only detects the uronic acid content which is present in both chondroitin 4 and 6-sulfate, but not in keratan sulfate. In order to investigate the true nature of disease, the composition of different species in facet cartilage should be demonstrated in future although its significance in the pathogenesis of osteoarthritis is not known yet.

APPENDIX 1

Low back pain in Chinese industrial population

1. Aims:

to study the incidence of low back pain among the Chinese industrial population, and to study the relationship between the pain severity and the various causative factors (demographic and other work-related factors).

2. Materials and methods:

a. 200 workers had involved in the study of incidence of low back study while 400 workers in the study of the relationship between the pain severity and the various causative factors. They were all come from Paper Manufacturing Factory, Guangzhou, People's Republic of China.

b. Standard questionnaire was used to ask for 60 questions. These included demographic data, functional disability which determined by a modified "Oswestry's pain assessment protocol", work-relating factors and etc.

c. Statistical method 'cluster analysis' was used to stratify the severity of symptoms into mild, moderate and severe 3 categories. The grading was based on the "Oswestry's pain assessment protocol". Then the 'k nearest neighbour' method was utilized to test the correlation between the original 3 categories and the "predicted" 3 categories as determining by those causative factors.

3. Results:

A. Incidence of low back pain among those 200 workers was as high as 74%.

B. Factors which had no correlation with pain severity were:

1. Weight
2. Education
3. Smoking
4. Family history
5. Past injury, illness or operation
6. Past injury cause or location
7. Year in job
8. Present post
9. Working hour or shifting hour
10. Squatting, bending, weight lifting frequency during working hour.
11. Contact with vibration equipment
12. Low back pain instruction received previously
13. Work injury

C. The following factors had no significantly correlation with the pain severity, but some correlation was observed after statistical analysis.

1. Sex
2. Age
3. Height
4. Sport frequency
5. Standing, sitting hour during work
6. Other system illness
7. Type and place of work

APPENDIX 2

Standardized back clinic assessment sheets

BACK CLINIC, P.W.H.

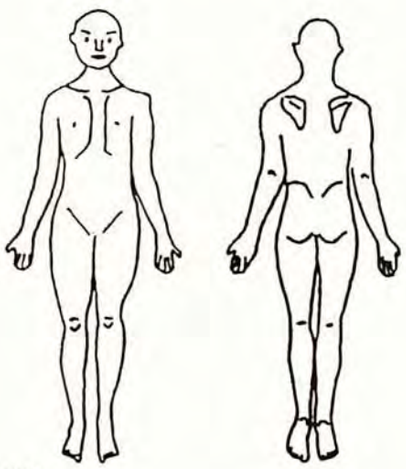
Name :	Sex/Age :	HKID No.:
O & T No.:	Physio. No.:	X-ray No.:

Occupation : MAB ☐

Past medical history :

Onset. medical Rx & response :

Physio. Rx & response :



Agg. act.

Eas. act.

Date :

Date :

Date :

Date :

Date :

Date :

Date :

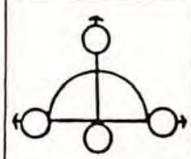
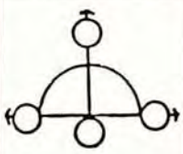
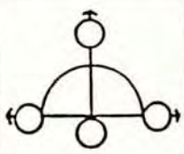
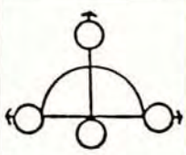
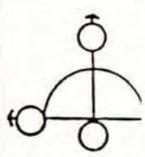
Date :

OrSD/27a(3/87)

OTSD/27b(3/87)

Name :

H.N. No.:
Physio. No.:

Date					
Back Pain					
Sciatica					
Impulse Pain					
Sitting Pain					
Spinal Claudication					
Functional Score					
Others					
Local Tenderness					
SLR R / L					
Range of Movement					
Neurology					
Others					
X-ray					
Diagnosis					

Physiotherapist's Remarks				
Signature / Date				
Medical Officer's Reply & Treatment Recommended				
Signature / Date				

APPENDIX 3

Enzymatic method for the comparison of extraction of uronic acid from standard chondroitin 6-sulfate

1. Aim: to establish the consistency of my present modified method for the extraction of uronic acid from the cartilage.

2. Procedures:

(A). Reagents:

(a). Standard chondroitin 6-sulfate

(b). Phosphate buffer (0.05 mol/l, PH 7.0)

2.7305 g potassium di-hydrogen phosphate(KH_2PO_4) and 2.8356 g di-sodium hydrogen phosphate(Na_2HPO_4) were dissolved in 100 ml deionized distilled water.

(c). Potassium acetate (20%)

2 g potassium acetate was dissolved in 10 ml deionized distilled water.

(B). Extraction of uronic acid from the standard:

Various concentrations of chondroitin 6-sulfate were digested in 5 ml of phosphate buffer, containing 2 mg per ml crystalline trypsin (Sigma, USA.) for 16-20 hours at 37^{o}C and then homogenization in a Kinematica (Polytron PTA 10S, Switzerland.) for 1 minute. Four ml of supernatant was brought to 40% ethanol by the addition of 4 ml of ethanol and 1.6

ml of aqueous 20% potassium acetate. After standing at 4°C overnight, the precipitate was removed by centrifugation and the supernatant brought to 50% ethanol concentration, allow to stand overnight at 4°C, and centrifuged. A third precipitate was collected in similar fashion by bringing the ethanol concentration in the supernatant to 75%. Each precipitate was lyophilized for at least 6 hours. The first precipitate was dissolved in 1 ml of deionized distilled water, the second and third was dissolved in 1 ml water, and portions were used for determination of uronic acid.

(C). Detection of uronic acid:

The same procedures as my present method.

3. Results:

Figure 17 indicates that the present method is superior for the extraction of uronic acid from the standard chondroitin6-sulfate. P value < 0.001.

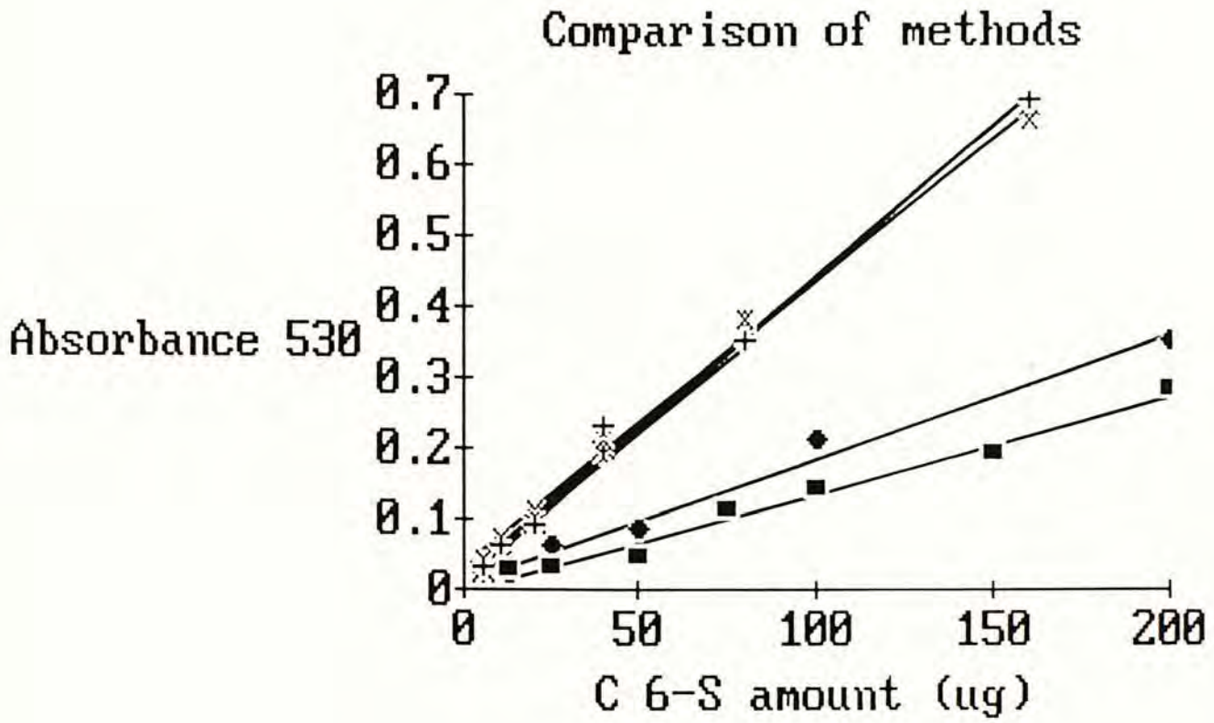


Figure 17: Results comparison between the methods for the extraction of uronic acid content from the standard chondroitin 6-sulfate.
(x, +): the present method
( ): enzymatic method

APPENDIX 4

Stability of standard chondroitin 6-sulfate in various conditions

1. Aim: to test the consistency of uronic acid existence in the present modified method and in other conditions.

2. Procedures: standard chondroitin 6-sulfate was added to various conditions.

   (1). Standard was dissolved in deionized distilled water.

   (2). Standard was dissolved in deionized distilled water and heated at 90°C for 45 minutes.

   (3). Standard was dissolved in deionized water and added to 6 mol/l sulfuric acid. 50 μl standard in various concentration was added to 950 μl 6 mol/l sulfuric acid. Thus the dilution will have a mild decrease of concentration.

   (4). Standard in water was added to 6 mol/l sulfuric acid and heated at 90°C for 45 minutes.

Detection of uronic acid was the same as the previous one.

3. Results:

The yielding of uronic acid in the various conditions are shown in Figure 18.

There were no differences among the various conditions except in condition (2) and (3), the P value < 0.05, (2) gave the lowest sensitivity. As a result, the condition which using at the present was good enough to detect the uronic acid content.

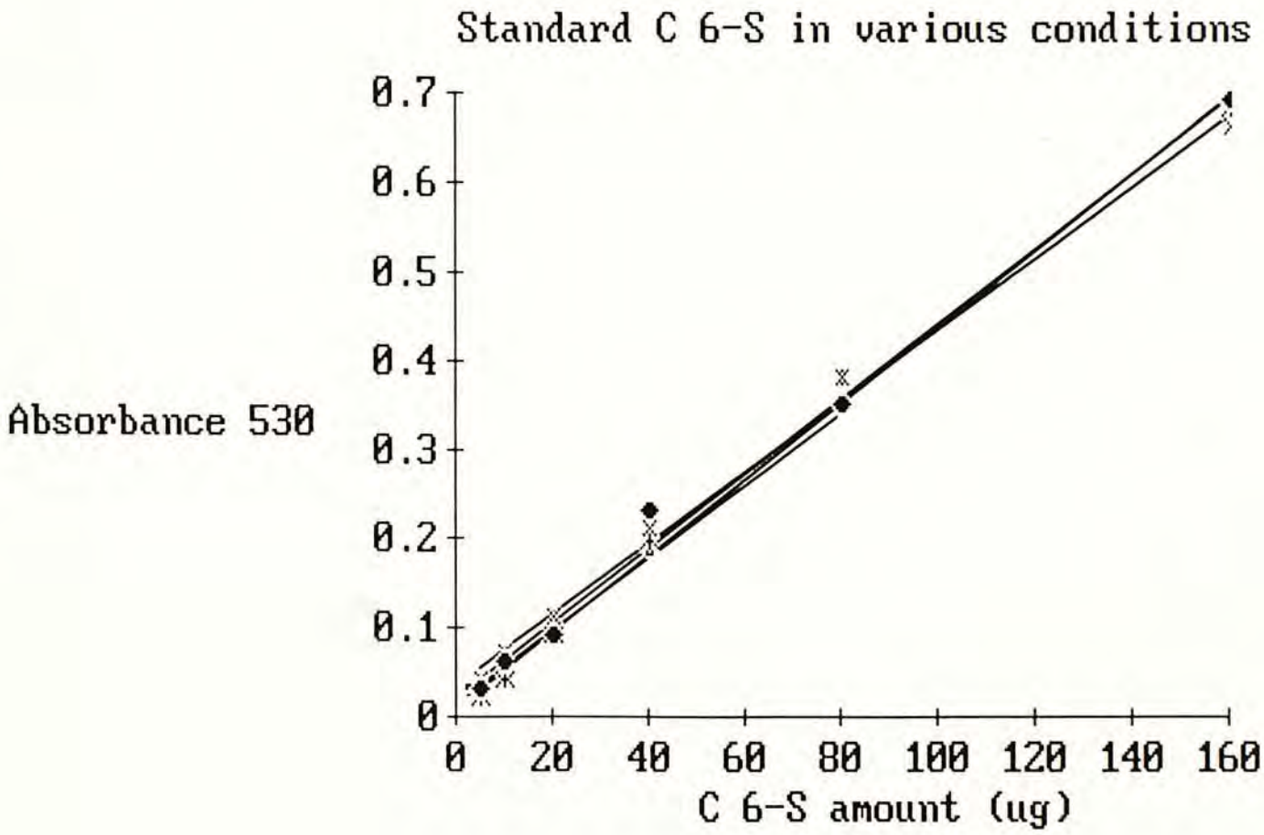


Figure 18: Stability of standard chondroitin 6-sulfate in various condition

APPENDIX 5

Original Data of "Gross Linear Index" (GLI) and "Histological Linear Index" (HLI)

No.	"GLI"	"HLI"
1	2.20	1.75
2	1.00	1.00
3	1.23	1.00
4	1.81	1.50
5	2.26	2.34
6	1.26	1.35
7	1.41	1.24
8	1.00	1.00
9	1.00	1.00
10	1.00	1.26
11	1.00	1.00
12	1.27	1.37
13	2.24	1.40
14	2.35	2.09
15	2.66	1.72
16	2.00	1.46
17	2.30	1.65
18	2.30	1.45
19	2.46	2.14
20	2.00	2.20
21	2.18	2.00
22	2.33	2.00
23	1.00	1.00
24	1.00	1.00
25	2.27	1.60
26	2.15	2.00
27	2.41	2.00
28	2.57	1.39
29	2.00	1.30
30	2.64	2.63
31	2.00	2.44
32	3.00	2.88
33	2.00	2.88
34	2.00	2.00
35	1.42	1.67
36	1.50	1.55
37	1.60	2.05
38	1.00	1.22
39	1.00	1.30
40	1.66	1.63
Mean:	1.81	1.66
SD:	0.59	0.52

APPENDIX 6

Original Data of Water Content of Various Gradings

No.	Grade I	Grade II	Grade III
1	66.0	70.4	67.4
2	65.8	76.2	74.1
3	64.5	73.0	73.2
4	72.0	70.0	69.0
5	68.4	74.0	72.0
6	69.0	73.3	75.0
7	60.0	68.8	52.9
8	65.1	74.7	68.6
9	69.7	69.6	
10	64.7	68.3	
11	67.6	74.1	
12	68.4	74.4	
13	66.9	72.8	
14	67.5	74.1	
15	64.0	74.7	
16	67.4	74.6	
17	64.4	74.2	
18	67.1	68.5	
19	66.9	73.9	
20	71.4	72.3	
21	67.6	70.0	
22	62.5	68.8	
23	68.0	71.8	
24	68.8	71.6	
25	68.6	74.4	
26	68.4	74.2	
27	64.3	71.8	
28	65.3	74.1	
29	63.4	71.6	
30	63.8	70.0	
31	71.4	69.5	
32	70.7	58.9	
33	68.8	68.4	
34	69.1	74.1	
35	62.1	69.4	
36	60.2	72.3	
37	70.9	71.2	
38	70.6	68.5	
39	72.0	66.9	
40	72.4	70.0	
41	73.7	74.2	
42	71.6	71.3	
43	72.5	70.4	
44	70.4	73.0	
45	78.0	73.5	
46	72.0	70.2	
47	67.0	70.7	
48	64.0	71.2	
49		74.0	
50		66.5	
51		65.0	
Mean:	67.8	71.4	69.03
SD:	3.66	3.08	7.08

APPENDIX 7

Original Data of Uronic Acid Content of Various Gradings

No.	Grade I	Grade II	Grade III
1	72.7	87.4	58.3
2	94.5	79.4	59.5
3	82.7	66.6	58.1
4	91.4	48.7	43.0
5	96.7	92.1	49.0
6	99.8	102.6	55.0
7	78.3	83.8	31.2
8	103.9	80.9	84.3
9	100.8	58.5	77.3
10	151.8	54.5	84.3
11	172.4	86.8	82.9
12	84.3	77.2	31.0
13	89.8	49.2	68.0
14	76.6	85.4	47.3
15	86.4	43.7	48.6
16	64.7	67.2	43.5
17	91.1	51.2	25.2
18	54.0	74.4	25.8
19	80.3	121.6	29.4
20	94.0	97.1	28.8
21	87.0	71.7	59.6
22	84.7	50.6	33.9
23	68.3	69.6	
24	64.0	60.8	
25	67.9	76.6	
26	80.1	73.3	
27	58.0	119.9	
28	47.5	99.7	
29	70.3	92.4	
30	87.4	77.8	
31	95.6	99.3	
32	67.1	85.6	
33	105.4	93.4	
34	73.1		
35	97.7		
36	76.3		
37	82.4		
Mean:	85.9	78.2	51.0
SD:	23.3	19.7	19.5

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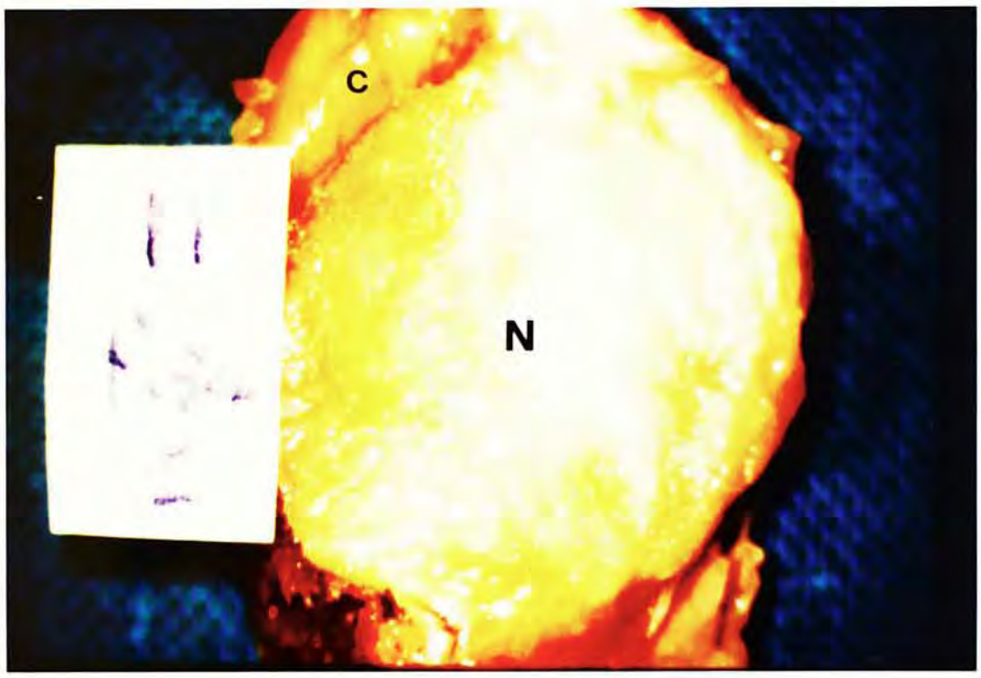

Figure 8. Gross appearance of normal facet articular cartilage('N'). This is a right side L34 level inferior facet surface. The whole articular surface of this facet cartilage was classified abnormal, that is, smooth and glistening surface and firm nature.'C' represents capsule of the facet joint.

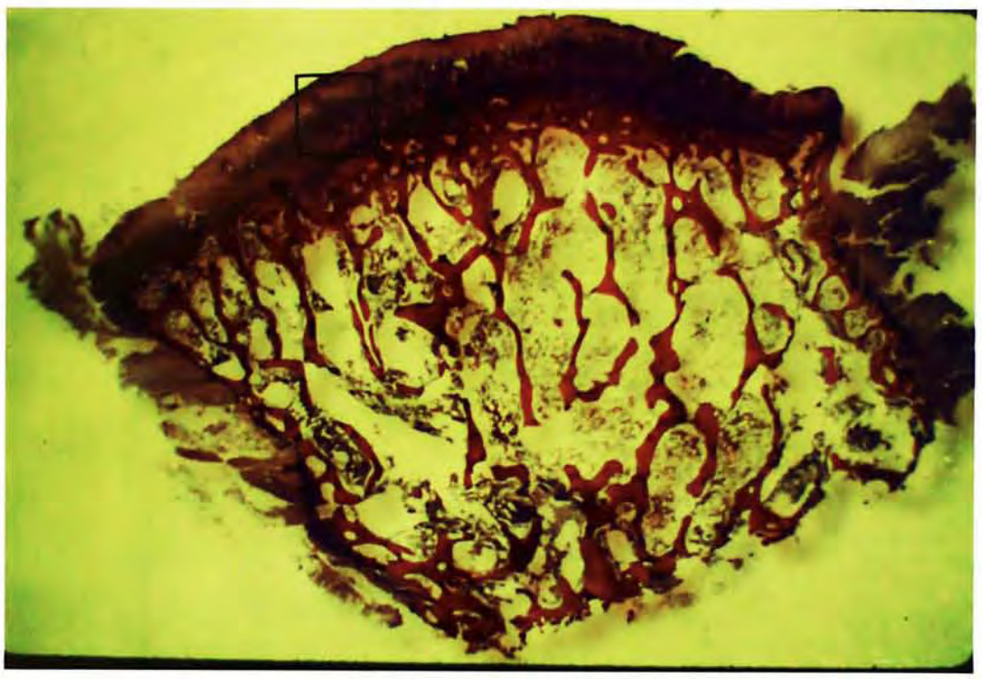

Figure 9. Vertical sectioned slide of normal facet joint. The intact articular surface and normal appearance of different zones. Higher magnification was proceeded for the 'square' area.

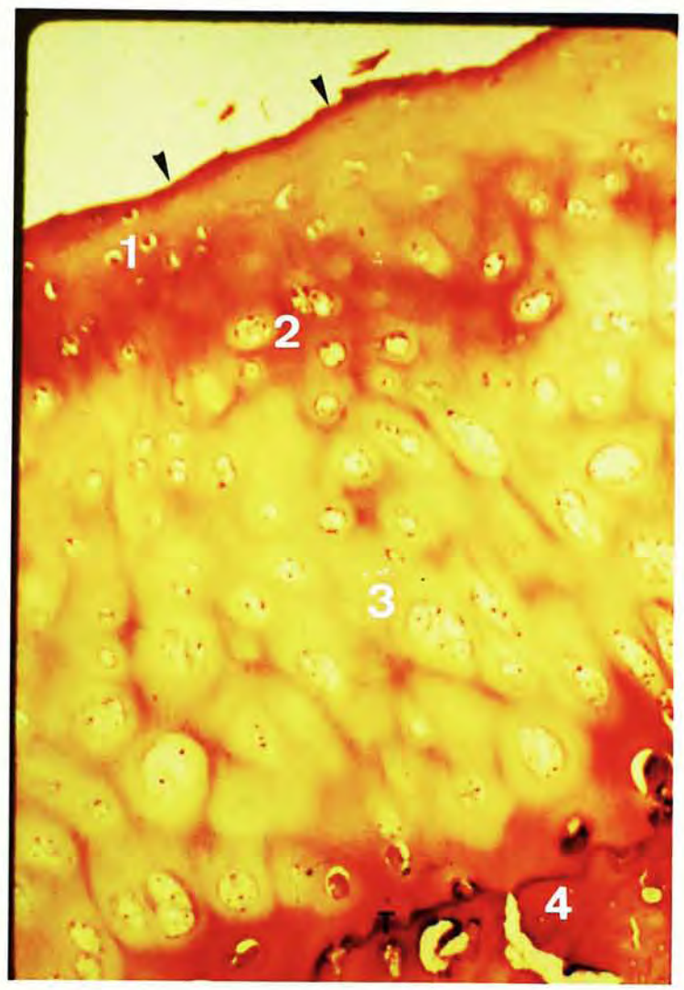

Figure 10. Histological appearance of normal facet articular cartilage (x 6.3). Normal appearance of 4 zones were identified,'1': superficial zone, '2': tangential zone, '3': radial zone, '4': calcified zone. The intact surface is indicated by the 'arrow' marker. 'T' is the tidemark of the cartilage, which is clearly shown.

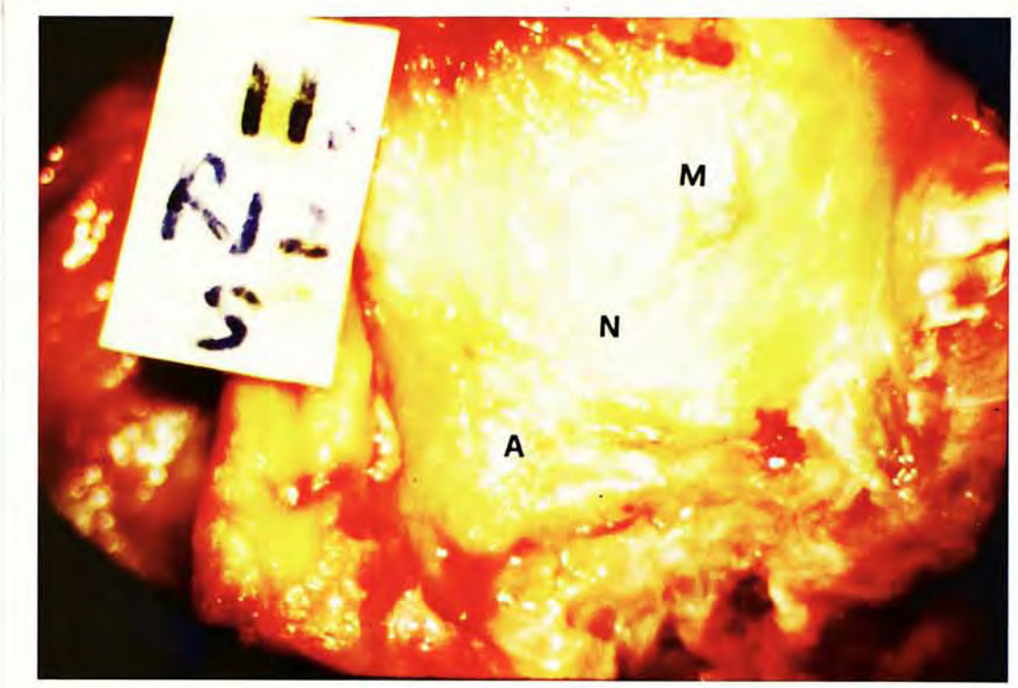

Figure 11. Gross appearance of facet joint cartilage with different degree of degeneration (I). This is a right side L12 level superior facet surface. 'M': moderate degree of degeneration with rough, softening and thinning articular surface, but no subchondral bone was exposed. 'N': normal appearance. 'A': advanced degree of degeneration with marked erosion and only thin layer of soft and irregular surface.

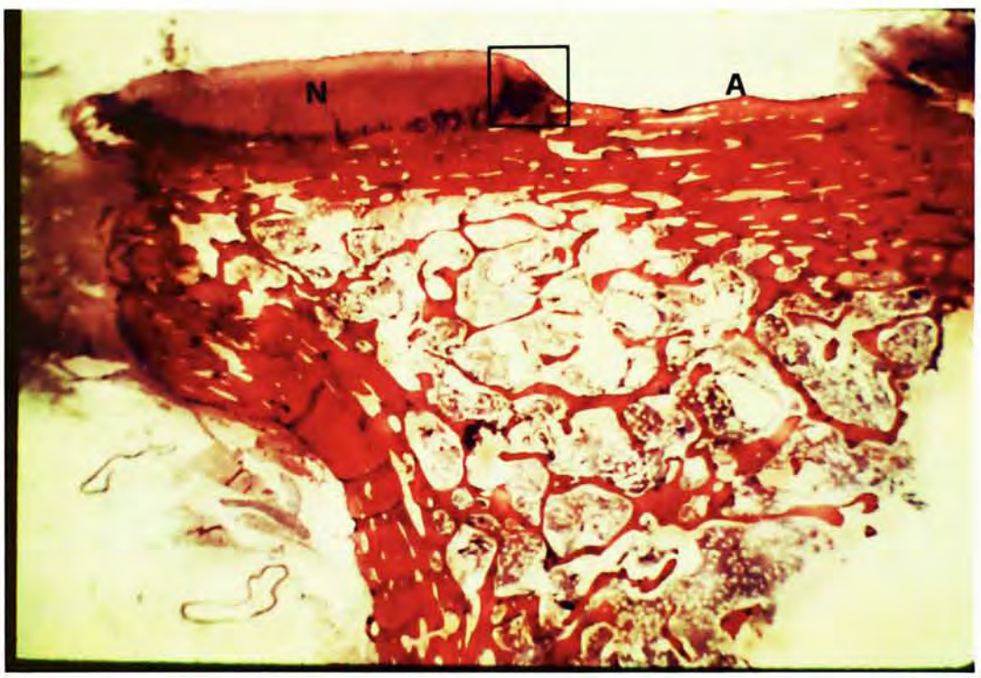

Figure 12. Vertical sectioned slide of normal and advanced degenerative facet joint cartilage. 'A': advanced degree of degeneration with loss of articular surface. 'N': normal cartilage. Higher magnification was proceeded for the 'square' area.

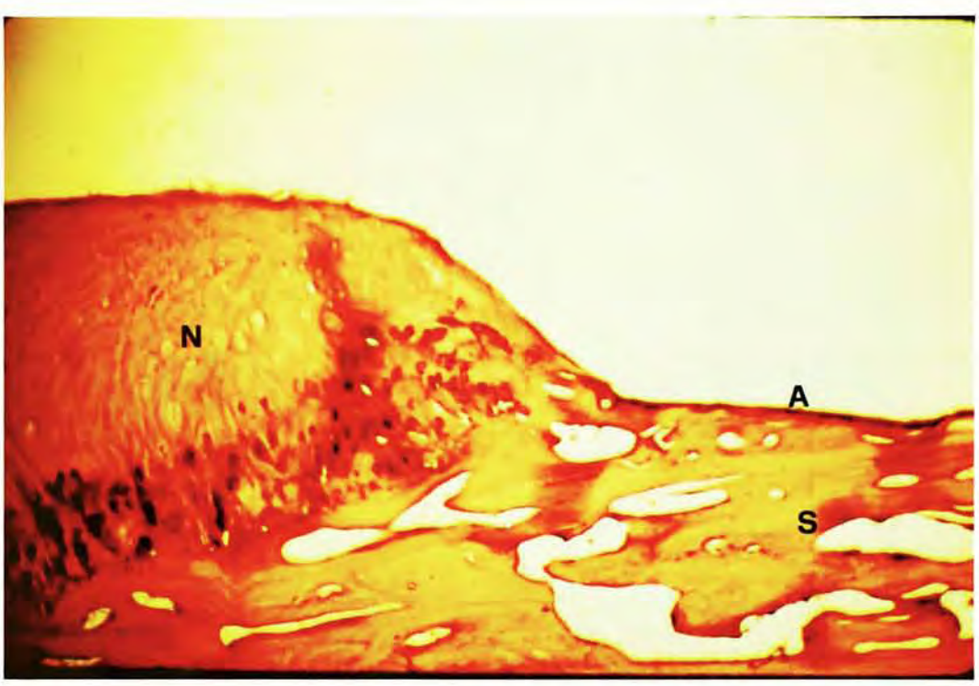

Figure 13. Histological appearance of normal and advanced degenerative facet joint cartilage (x 2.5). Advanced degree of degeneration was indicated by loss of cartilage and thickened subchondral bone ('S').

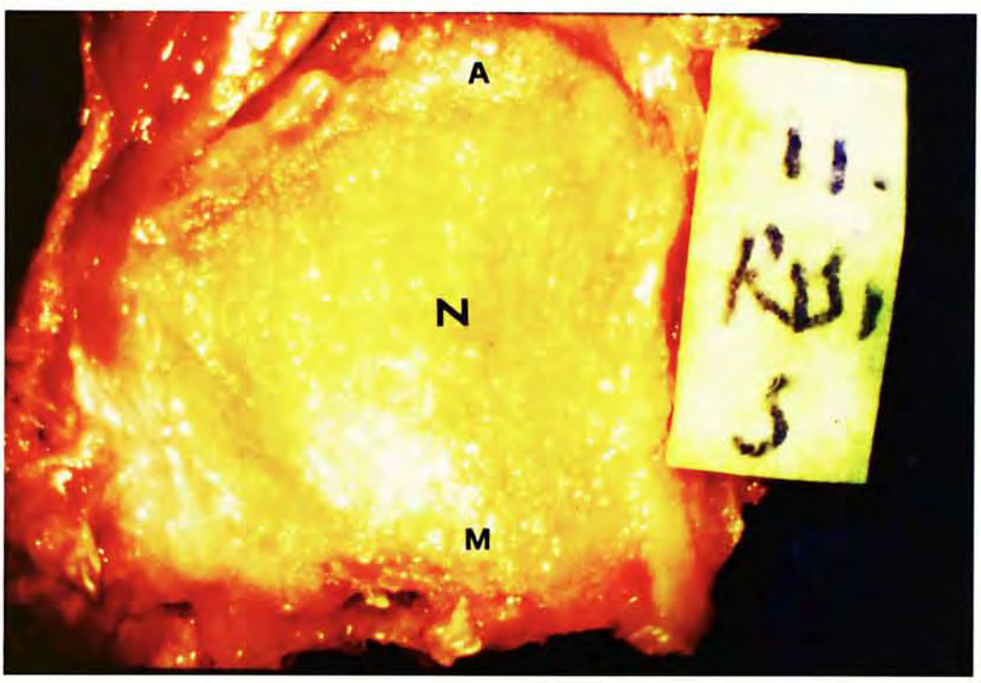

Figure 14. Gross appearance of facet joint cartilage with different degree of degeneration (II). This is a right side L5S1 level superior facet surface. This surface shows advanced degree of degeneration ('A'), normal cartilage ('N') and moderate degree of degeneration ('M').

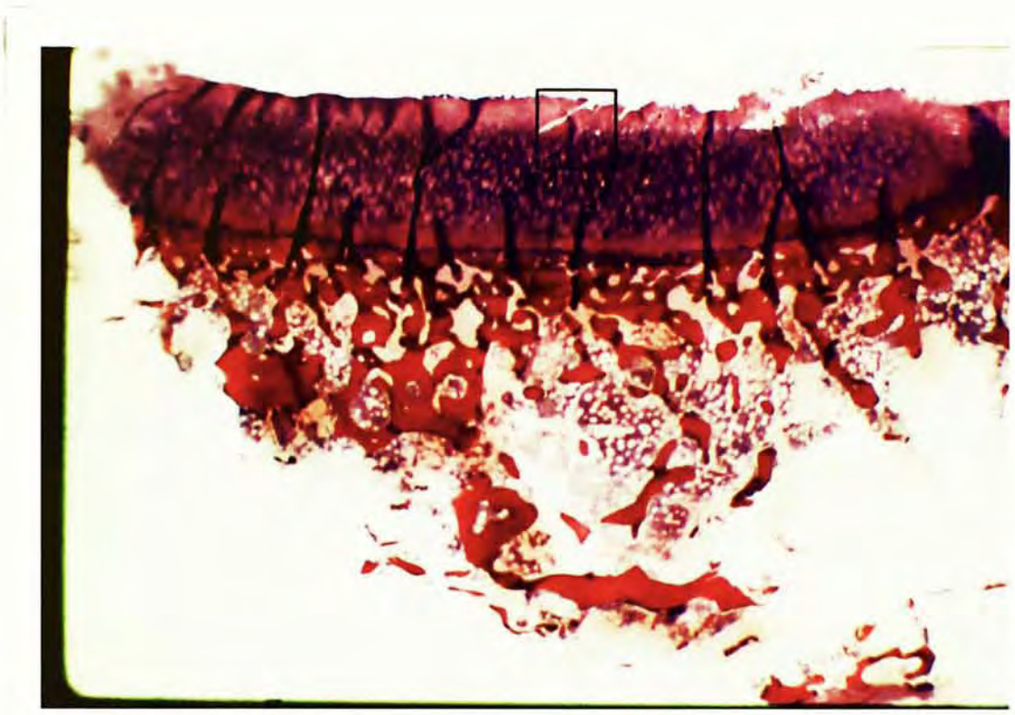

Figure 15.Vertical sectioned slide of normal and moderate degenerative facet joint cartilage . Higher magnification was proceeded for the 'square area'.

Figure 16. Histogical appearance of moderate degenerative facet joint cartilage (x 6.3). Irregular surface, fibrillation of the cartilage (arrow without tail), cluster of cartilage cell (arrow with tail) were typical features of degenerative change of cartilage.